AN ANALYTICAL STUDY OF LIVER ABSCESS

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CERTIFICATE

This is to certify that this dissertation titled **"AN ANALYTICAL STUDY OF LIVER ABSCESS"** submitted by **DR.R.G.CHANDRA MOULI** to the faculty of General Surgery, The TamilNadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MS degree Branch I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from 2009 to 2011.

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CONTENTS

INTRODUCTION

As we know, the benign conditions of liver have lots of clinical implications. Of these, liver abscess is a common condition worldwide particularly in the tropical countries. Among the developing countries, India has 2nd highest incidence of liver abscess in the world. There are many pre disposing factors for liver abscess. The infective agents causing liver abscess can be classified as bacterial, parasitic or fungal causes. Among all, pyogenic abscesses accounts for four fifth of liver abscess in developed countries, whereas amoebic liver abscess account for two third of liver abscess in developing countries.

Entemoeba histolytica infection affects 10% of world population , pyogenic liver abscess affects 30/1,00,000 hospital admissions and the sex incidence is almost equal .

The management of amoebic liver abscess evolved with recognition of colonic amebiasis as the antecedent source of liver abscess. Early treatment with open surgical drainage alone had met with limited success . Efforts to treat both liver abscess and colonic infestation improved the success rate.

With the development of systemic amoebicidal agents along with USG guided closed aspiration became the treatment of choice. The present laparoscopic era has narrowed down open procedure .

AIMS AND OBJECTIVES

1. To study the clinical presentation of liver abscesses i.e. Distribution with respect to age and sex, mode of presentations.
2. To study the risk factors associated with liver abscess .
3. To study the microbiological diversity in liver abscess .
4. To study the effectiveness of various treatment modalities for liver abscess .
5. To study the effectiveness of different modes of management.

REVIEW OF LITERATURE

Historical aspects

The history of amoebiasis goes back to the era of Indian great Susruta (600 BC) who gave description of amoebic dysentery as Athisara, incriminating the germination of parasites in the intestines by drinking impure water.

Liver abscess was first drained in the Hippocratic era, and master of medicine successfully practiced the draining of pus.

The parasite was first described by Lambi and was demonstrated in pus from the tissues adjoining the liver abscess by the microbiologist Koch. Councilman and Lafleur (1891) in Baltimore proved the clinical and pathological evidence that amoeba was responsible for liver abscess.

Roger (1918) in his famous paper described “The protozoal organism reaches the liver by portal circulation and they entangle in the interlobular veins producing congestion of liver, he established that amoebae are constantly present in the walls of the abscess though not frequently in pus.”

SURGICAL ANATOMY

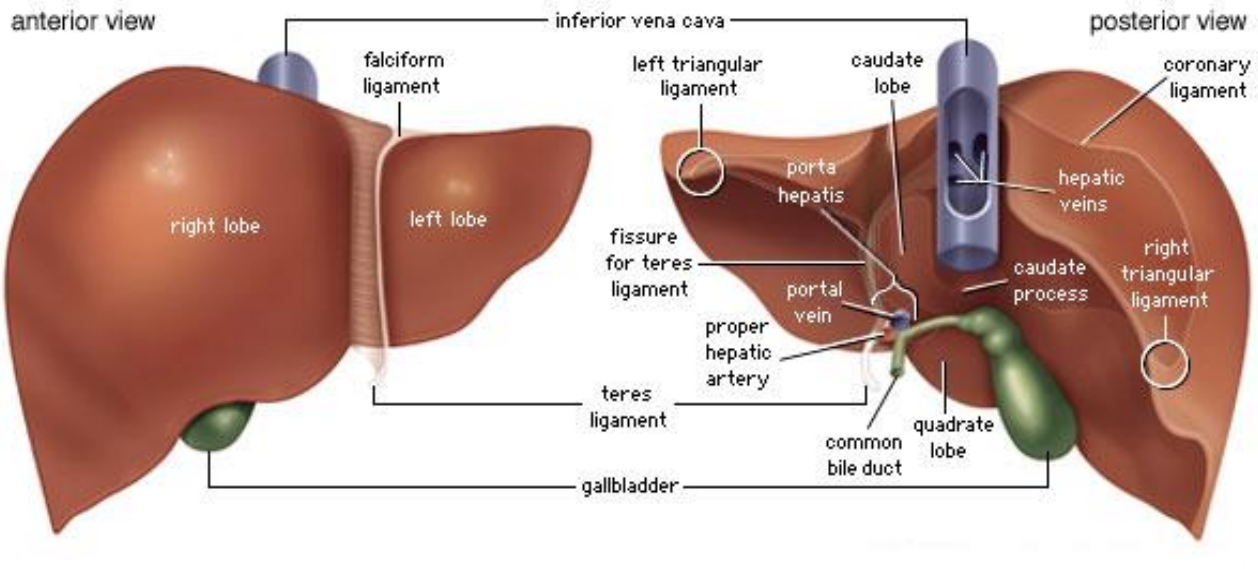
The liver, the largest gland in the body weighs approximately 1500g and receives about 1800 ml of blood /min. This wedge shaped organ occupies most of the right hypochondrium and epigastrium. It has two surfaces, visceral and diaphragmatic.

The diaphragmatic surface, convex is divided into anterior, posterior and right surfaces. Sharp inferior border separates right and anterior surface from visceral surface.

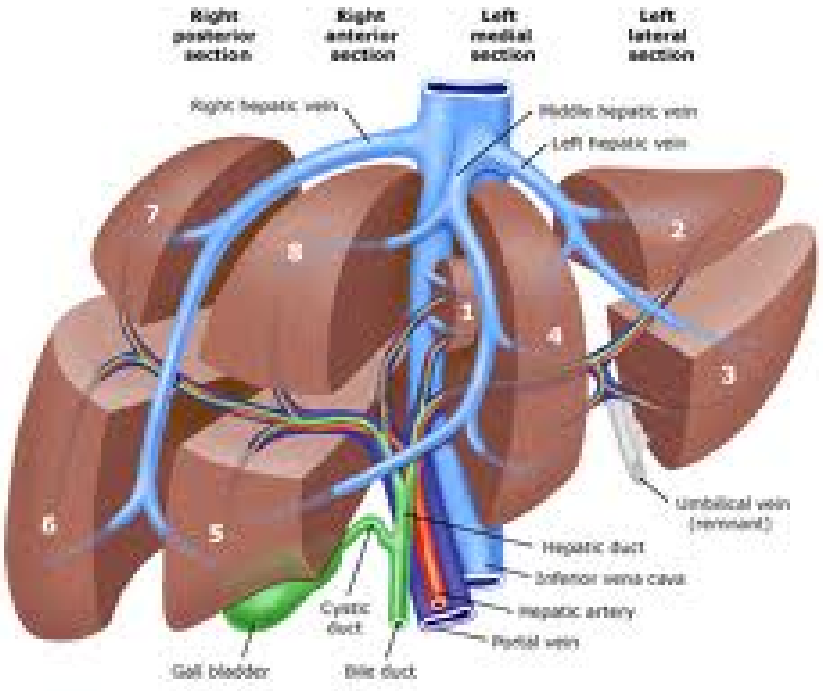
The main vessels and ducts enter or leave at the porta hepatis which is on the visceral surface, but hepatic vein emerges from the diaphragmatic surface.

The inferior border is notched by the ligamentum teres. The falciform ligament ascends on the anterior surface to reach the superior surface where a reduplication of the left leaf forms the left triangular ligament. The right leaf becomes the upper layer of the coronary ligament.

The venacava lies in the deep groove on the posterior surface. To the right is the triangular bare area, with the vena cava at its base and with sides formed by superior and inferior layers of coronary ligament. The apex where these two layers meet is the right triangular ligament.



Surgical Anatomy of Liver – Diaphragmatic surface and visceral surface



Couinaud's functional segments of Liver

At the porta hepatis, lie the hepatic ducts, hepatic artery and portal vein. They lie in the order vein-artery-duct, with the duct in front. There are also nodes and nerves of the liver. The bare area is in contact with the diaphragm and right suprarenal gland.

The visceral surface is related to stomach, duodenum, hepatic flexure of colon and right kidney.

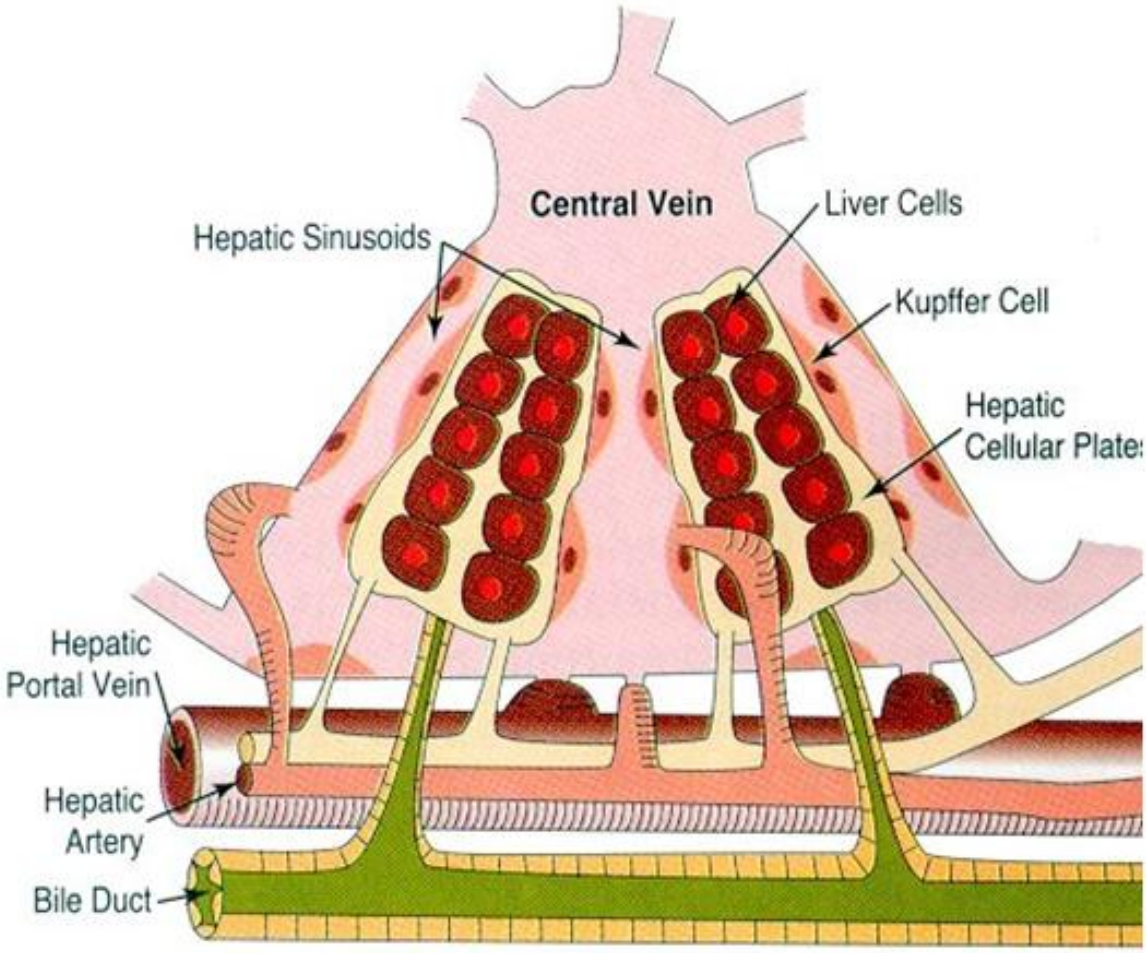
Anatomically the liver has two lobes, right and left divided by the falciform ligament anteriorly. The right lobe is further divided into right lobe proper, caudate and quadrate lobes. The caudate lobe lies in between IVC and fissure for ligamentum venosum. The quadrate lobe lies in the visceral surface between the gallbladder fossa and fissure for ligamentum teres.

SEGMENTS OF LIVER

On the basis of blood supply and biliary drainage there are four main hepatic sectors: left lateral, left medial, right anterior and right posterior .These four sectors further subdivided into eight segments.

SEGMENT 1 - caudate lobe - An autonomous segment receiving blood from right and left branches of hepatic artery and portal vein, draining bile

ANATOMY OF A LIVER LOBULE



into right and left hepatic ducts and having independent venous drainage into 1VC.

The left lateral sector contains segment Il posteriorly, segment Ill anteriorly with the left hepatic vein between them.

Segment IV is recognized on the visceral surface as the quadratelobe. Segment V and VI are the inferior segments of the right anterior and posterior sectors respectively. Segments VII and VIII are the superior segments of the right posterior and anterior sectors respectively.

HISTOLOGY

Liver is seen to be composed of parenchymal cells arranged in anastomosing and branching plates which form a three dimensional lattice. Plates of parenchymal cells radiate from central vein like spokes of a wheel, each being 1 mm in diameter. Portal triad or portal areas or portal canal contains a branch of portal vein, a branch of hepatic artery and an interlobular bile ductule.

In humans, liver contains 3-6 portal canals per lobule. Between parenchymal plates are sinusoidal blood spaces. Sinusoids are irregularly disposed, normally in a direction perpendicular to the lines connecting central veins. Walls of the sinusoids consist of endothelial cells called Kupffer cells. Potential spaces between hepatic cells and walls of sinusoids

are called space of Disse. This space is continuous with larger space that surrounds the portal areas known as the space of Moll.

The continuous liver tissue is pervaded by two systems of tunnels, the portal tracts and hepatic canals, which are arranged in such a way that they do not meet each other. As far as possible two systems run in planes perpendicular to each other. The terminal branches of portal vein discharge their blood into sinusoids.

PYOGENIC LIVER ABSCESS:

Incidence:

Pitt and Zuidema, in 1973, documented an admission prevalence at the Johns Hopkins Hospital, Baltimore of 13 per 1,00,000 A more recent publication from that same institution comparing a pair of 21-year time periods suggests that the incidence of pyogenic hepatic abscesses has increased significantly to 20 per 1,00,000 admissions[(1)]. In a series from Duke University Medical center, the incidence over period 1979 to 1986 was 22 cases per 1,00,000 hospital admissions. This compared with a figure of 11.5 cases per 1,00,000 admission during the period 1970 to 1978. A further series from university of California San Francisco Hospital describes an incidence of 22 cases per 100000 hospital admissions. In each of these series, the peak incidence occurred early in sixth decade thereby

suggesting a shift in age. In two of these series, the most common cause of hepatic abscess was found to be cryptogenic in that no obvious predisposing cause was identified. It remains possible that there has been a recent true increase in the incidence of primary cryptogenic PLA.

Etiology and Pathogenesis:

Most pyogenic liver abscesses are caused by infection in biliary or intestinal tracts. As a result, the causes of liver abscesses have been divided into six categories based on the route of extension of infection.

1. Biliary- from ascending cholangitis.
2. Portal vein- as in pyelophlebitis resulting from appendicitis or diverticulitis.
3. Hepatic artery- from septicemia.
4. Direct extension- from contiguous disease process
5. Traumatic- from blunt or penetrating injuries
6. Cryptogenic- when no primary source of infection is found even after abdominal exploration of autopsy.

In the 1975 report by Pitt and Zuidema from the Johns Hopkins Hospital, 51% of the patients had a hepatobiliary or pancreatic neoplasm (23%) or a benign biliary tract condition (28%) [1]. However in the more recent series from Johns Hopkins, the underlying problem was a malignant

disease in 42% of patients. Similar trends have been reported by Branum and Associates at Duke University in North Carolina.

Analysis of abscesses with a portal cause reveals that several other intra abdominal disease process have replaced appendicitis as the leading cause in this category. At present, the frequent sources of portal vein sepsis resulting in liver abscess include diverticulitis, perforated ulcers, and perforated carcinomas. The relative incidence of pyogenic liver abscesses resulting from systemic bacteremia, direct extension, and trauma have remained relatively constant since 1950.

The pathogenesis of cryptogenic abscesses is still uncertain, although several theories have been proposed. In 1972, Lee and Block noted an increased incidence of anaerobic infections in their patients. They suggested that cryptogenic abscesses may develop from small areas of intrahepatic thromboembolim or infarction that become infected secondarily by anaerobic bacteria was present in 45% of all liver abscesses, the most commonly encountered organisms were anaerobic and microaerophilic streptococci, bacteroides fragilis and fusobacterium.

Patients with compromised host defenses have an increased risk of developing pyogenic liver abscesses. Diabetes mellitus was present in 15% of the patients of Altemeier et al, and liver abscesses have been found in

children with leukemia, chronic granulomatous disease, AIDS and other immunodeficiency disorders.

Pathology:

Generally, portal, traumatic, and cryptogenic liver abscesses are solitary and large, while biliary and arterial abscesses are multiple and small. If the primary lesion is located within the portal circulation, usually the' abscesses are large, single or multiple and in most cases confined to the right lobe of liver, the left lobe is rarely affected.

Kinney and Ferrebee' in a study based on experiments of serege in 1901[18], showed that there is a separate flow of blood from superior mesenteric vein to the right lobe of the liver, and from splenic vein to the left lobe of the liver[3] . This explains the preferential location of portal liver abscesses in the right lobe, which drains the intestines. Liver abscesses in both lobes will occur when the portal vein is filled with a septic thrombus.

In a review by Gyorff' and colleagues, 40% of the liver abscesses were found to be 1.5 to 5 cm in diameter, 40% were 5 to 8 cm, and 20% were 8 cm or larger[4]. Pyogenic liver abscesses localize to the right hepatic lobe in 65% cases, with the majority of these being solitary. The left lobe is solely involved in 12% of cases, with 23% of patients having bilateral abscesses. Bilateral disease occurs in 90% of cases with a biliary or arterial

source of infection distributing along the terminal branches of the portal triad. Fungal hepatic abscesses are most often multiple bilateral, and miliary in nature.

Microbiology:

On older literary evidence, sterile culture was found in up to 60% of all cases. Even in more recent reviews, the number of sterile abscesses still exceeds 10%. This low number is probably the result of inadequate anaerobic culture techniques. In a systematic search, Sabbaj & Co-workers found that anaerobic bacteria were present in 45% of all liver abscesses, the most commonly encountered organisms were anaerobic and microaerophilic streptococci, Bacteroides fragilis, and fusobacterium[5].

The observations of Onderdonk et al that the presence of anaerobic bacteria is necessary to produce intraperitoneal abscesses, it is likely that anaerobic bacteria are involved in a large percentage of hepatic abscesses [6]. It seems, that anaerobic bacteria are prominent in abscesses, secondary to hepatic tumors, and that the bacteriology of abscesses originating from lesions within the portal circulation closely resembles that of intraperitoneal infections, that is E. Coli, Enterococcus and Bacteroides.

Most tuberculosis lesions of the liver are miliary granulomas. Abscess like masses (Tuberculomas) sometimes form and spread along the

walls of the intrahepatic bile ducts (tuberculous cholangitis). Diagnosis may be difficult because both caseation and acid fast organisms can be absent. Tuberculomas are often, but not always, accompanied by an abdominal focus.

Miliary abscesses have been found in cases of disseminated granuloma inguinale. Hepatic clostridial infections cause gas abscesses, but most of the jaundice in disseminated infections is hemolytic.

Clinical Features

Most patients with pyogenic liver abscesses present with symptoms of less than 2 weeks duration. The most common presenting symptom is fever, which is noted in approximately 90% of patients. Pain is the next common symptom. Chills and weight loss occur in approximately one half of the patients, other symptoms like jaundice, diarrhoea, cough, anorexia can also be present.

The most common physical sign is an enlarged tender liver, which is found in 55% patients with pyogenic abscess. Jaundice is also found on physical examination in approximately one half of the patients. Chest symptoms and physical findings are found in approximately one fourth of the patients. Abdominal examination reveals a palpable mass or ascites in about 25% of patients where as splenomegaly is detected in only 10%.

Almost all patients with pyogenic liver abscess have abnormal hematological and liver function tests. Leucocytosis is noted in approximately 70 to 90% of patients. Many patients are also found to be anaemic and this usually reflects the presence of chronic disease or a prolonged sub acute presentation. Erythrocyte sedimentation rate is also elevated. The most frequent liver function test abnormality observed in patients with hepatic abscess is an elevated alkaline phosphatase. This is seen in approximately 80 -90% of patients. Bilirubin is elevated in 40-60% of patients. Transaminases are also abnormal. Hypoalbuminemia is observed in approximately 70% of patients and mild elevation of prothrombin time is also frequently seen.

Radiological investigations:

Chest radiographs are abnormal in approximately 50% of patients presenting with hepatic abscesses. Changes suggestive of sub-diaphragmatic pathology include an elevated right hemi diaphragm; a right pleural effusion and right lower lobe atelectasis. Similar findings are occasionally found in the left thoracic cavity if the abscess involves the left hepatic lobe.

If gas-forming organisms are present within the abscess and air fluid level may be seen. Air within an unoperated biliary tree may also be demonstrated, confirming the diagnosis of cholangitis. Rarely portal venous gas may be seen on an abdominal x-ray, confirming pyelophlebitis. Portal venous

gas appears as branching linear lucencies along the peripheral portion of the liver. Air within the biliary tree tends to be seen more centrally. However, it is rarely possible to make this distinction on the basis of plain film appearances alone and US or CT are usually required.

With the increased incidence of biliary causes of pyogenic liver abscess cholangiography has become more important in the diagnosis of many of these patients. Either ERCP or MRCP were helpful in defining biliary anatomy as well as in outlining the abscess cavities in approximately two third of the studies

Satiani and Davidson found liver scans to be positive in 90% of patients with a solitary abscess but in only 70% of patients with a multiple abscesses[7]. The decreased accuracy of liver scanning in patients with multiple abscesses is explained by the finding that abscesses smaller than 2cm are not detected by this technique. A liver scan with technetium-99 sulfur colloid will show the defect in over 80% of all cases. Other radiologic methods, such as scanning with indium Ill labelled leucocytes and gallium 67 are used.

Liver scans have provided a means for early detection of abscesses that was not previously available.

Barreda and Ros considered Ultrasonogram to be modality of choice in studying the internal nature of hepatic abscesses [8] .

The disadvantages are:

1. Cannot always visualize the liver dome and may miss lesions in this area
2. Multiple microscopic abscesses such as those generally found with ascending cholangitis, may not be appreciated.
3. Fatty infiltration may produce a markedly echogenic liver, with resulting failure to detect a small abscess.

USG and CT of abdomen have replaced liver scans as the method of choice for radiologic proof of hepatic abscesses. Abscesses that are large enough to be suitable for diagnostic aspiration or therapeutic drainage can be equally well diagnosed ultrasonography the lesion can be echogenic as well as nonechogenic. In the case of non-echoic lesions variable amounts of internal echo can be seen. Nearly all abscesses show distal sonic enhancement as long as no gas is present. Computed tomography may visualize hepatic collections as small as 0.5cm and CT may more easily identity multiple small abscesses. Most abscesses are inhomogeneous, but the density is generally lower than in the surrounding tissue. Intravenous administration of contrast material enhances the case by which abscesses can be diagnosed, and in a few cases the abscess cannot be detected until the contrast material has been administered. MRI has recently been used for the detection of hepatic abscesses.

TREATMENT

Once a diagnosis of hepatic abscess is suspected broad spectrum intravenous antibiotics should be started. Antibiotics therapy can be adjusted once the results of abscess cultures are available. Blood should also be sent for culture. Specimens should be cultured for acid —fast bacilli and fungi and this is particularly the case if there is a clinical suspicion of mycobacterium or fungal infections or if patients are immune suppressed. Empirical antibiotic therapy should include effective cover against aerobic gram-negative bacteria. Appropriate antibiotic combination would include ampicillin and aminoglycoside and metronidazole or a third generation cephalosporin such as cefotaxime together with ampicillin and metronidazole, alternatively a carbapenem antibiotic such as imipenem or meropenem may be used. Metronidazole will also be therapeutic for patients with amoebic liver abscess. All patients at risk of amoebic liver abscess should also undergo serological testing.

The duration of antibiotic treatment will vary according to the clinical setting. However antibiotic penetration into the abscess cavity is often poor and 2 weeks of intravenous antibiotics are usually recommended. Appropriate oral antibiotics are usually continued for a further 4 weeks.

In the pre-antibiotic era, untreated liver abscess was uniformly fatal. Following the publication of the review by Ochsner et al in 1938, surgical drainage was widely adopted and this resulted in dramatic reduction in mortality. Extra peritoneal drainage was recommended so as to avoid contamination of the peritoneal cavity. This was usually achieved via a posterior approach through the undersurface of the twelfth rib.

In 1985, Gerzof et al 27 published a series of 18 hepatic abscesses, 16 of which were successfully managed by percutaneous catheter drainage[9]. Only two patients required surgical drainage and no deaths were reported in this series. The following year Bertel et al 40 (1986) published a series 39 patients with pyogenic hepatic abscess; 23 patients were treated surgically, 16 patients underwent percutaneous drainage. Three of the percutaneously treated group required surgical drainage due to viscous abscess contents. However, the majority of patients were successfully treated. Mortality was 17% in the surgical group and 13% in percutaneoulsy-drained group [10]. Wong (1990) ‘“ described 21 patients with pyogenic liver abscess treated by percutaneous drainage[11]. This was successful in 85% of patients with mortality of less than 1 0%. Contraindications to catheter drainage include the presence of ascites, coagulopathy and proximity to vital structures.

Branum et al (1990) reported a series of 73 patients admitted between 1970 and 1986. During the period 1970 to 1978, 86% of patients (25 of 29) were initially treated by surgery. However, during the period 1971 to 1986, equal numbers of patients underwent surgery and percutaneous drainage as the first definitive therapy[12].

In 1996, Seeto and Rockey reported a series of 142 patients admitted between January 1979 and December 1994. During the first 3 years of this study 925 of patients (12 of 13) were initially treated by surgery. However, during the last 5 years of the study only I of 50 patients underwent surgery as the initial form of treatment. Percutaneous drainage was successful in 90% of these patients[13].

It has recently been suggested that percutaneous aspiration may be preferable to catheter drainage. The main advantages of needle aspiration over catheter drainage are the fact that it is less invasive, less expensive and needle aspiration avoids the problems related to follow-up catheter care or loss of catheter position. However, incomplete evacuation of the abscess cavity or rapid reaccumulation of abscess contents following percutaneous aspiration was considered indications for continuous catheter drainage. Percutaneous needle aspiration appeared to be less effective than PCD, though both procedures were shown to be safe with no major complications and no deaths.

Therapy with antibiotics alone may be appropriate in selected patients. For example, patients with malignant biliary obstruction and pyogenic liver abscess may be successfully managed with a combination of biliary decompression in these patients is invariably fatal irrespective of whether abscess drainage is performed or not[26].

It was initially thought that only patients with single PLA should be managed by percutaneous drainage with operative drainage reserved for patients with multiple or complex abscess. However, a number of authors have found that percutaneous treatments are equally effective for patients with both single and multiple pyogenic abscesses.

Liver resection is occasionally required for patients with pyogenic liver abscess. The indication for this is usually hepatolithiasis or intrahepatic biliary stricture. In other patients, hepatic destruction may be so severe that they are best served by liver resection. Clearly risks are involved as manipulation may produce a life-threatening bacteremia. it is therefore recommended that, following ligation of the vascular inflow, the involved hepatic vein should be ligated before parenchymal dissection is carried out.

Laparoscopic drainage is an attractive alternative for patients requiring open surgical drainage. The advantages of laparoscopic surgery in terms of reduced analgesia requirements, reduced morbidity, faster postoperative

recovery and shorter hospital stay compared to laparotomy are well documented. Laparoscopic US is also likely to be useful in this respect.

Outcome:

With the introduction of surgical drainage and systemic antibiotics mortality fell. In the 1980s, the widespread availability of US and CT facilitated earlier diagnosis and the development of percutaneous methods of drainage. This has resulted in a further fall in mortality. In the Johns Hopkins series, the overall mortality in the period 1952 to 1972 was 65% compared to a mortality of 31% during the period 1972 to 1993. Branum et al (1990) have reported a mortality of 19% between 1970 and 1986 [12] and most recently Seeto and Rockey (1996)6 have reported a mortality of 11% for patients presenting between 1979 and 1994 [13].

A number of studies have attempted to identify factors that are predictive of poor outcome. In a univariate analysis, Lee et al (1991)36 identified clinical jaundice, pleural effusion and bilobar abscesses to be risk factors for mortality were hypoalbuminemia, hyperbilirubinemia, elevated aspartate transferase and alkaline phosphatase and leukocytosis. Multivariate analysis revealed leukocytosis, hypoalbuminemia and pleural effusion to be independent risk factors for mortality. A multivariate analysis of 46 patients from Austria found

that a high bilirubin, low hemoglobin, and a high APACHE I score were predictors of a complicated clinical course or mortality[14].

Perforation of hepatic abscess was predictive of a complicated clinical course with a mortality of 3.07%. Chou et al (1994)48 also found that rupture of pyogenic liver abscess was associated with a significantly higher mortality (43.5% compared to 15.5%). In a considerably larger series of 384 patients, Chou et a (1994) identified age greater than 60, impaired renal function, hypoalbuminemia and elevated bilirubin to be independent risk factors of mortality [15].

The incidence of pyogenic liver abscess appears to be increasing. This is in part due to a more aggressive approach to the treatment of patients with hepatobiliary and pancreatic malignancy and the increasing use of cytotoxic drugs. Uncomplicated pyogenic liver abscess is now a disease with a good prognosis. This is illustrated by the fact that patients with cryptogenic liver abscess may have mortality as low as 2%.66 Factors such as delayed presentation and delayed diagnosis may both contribute to poor outcome. However, the major factor now contributing to mortality in patients with pyogenic liver abscess is the severity of the underlying disease and in particular the presence of malignancy.

AMOEBIC LIVER ABSCESS

EPIDEMIOLOGY

Infection with E. histolytica affects one tenth of the world's population and is considered responsible for at least 40,000 deaths annually, most infections occurring in the developing countries of the tropics and subtropics.

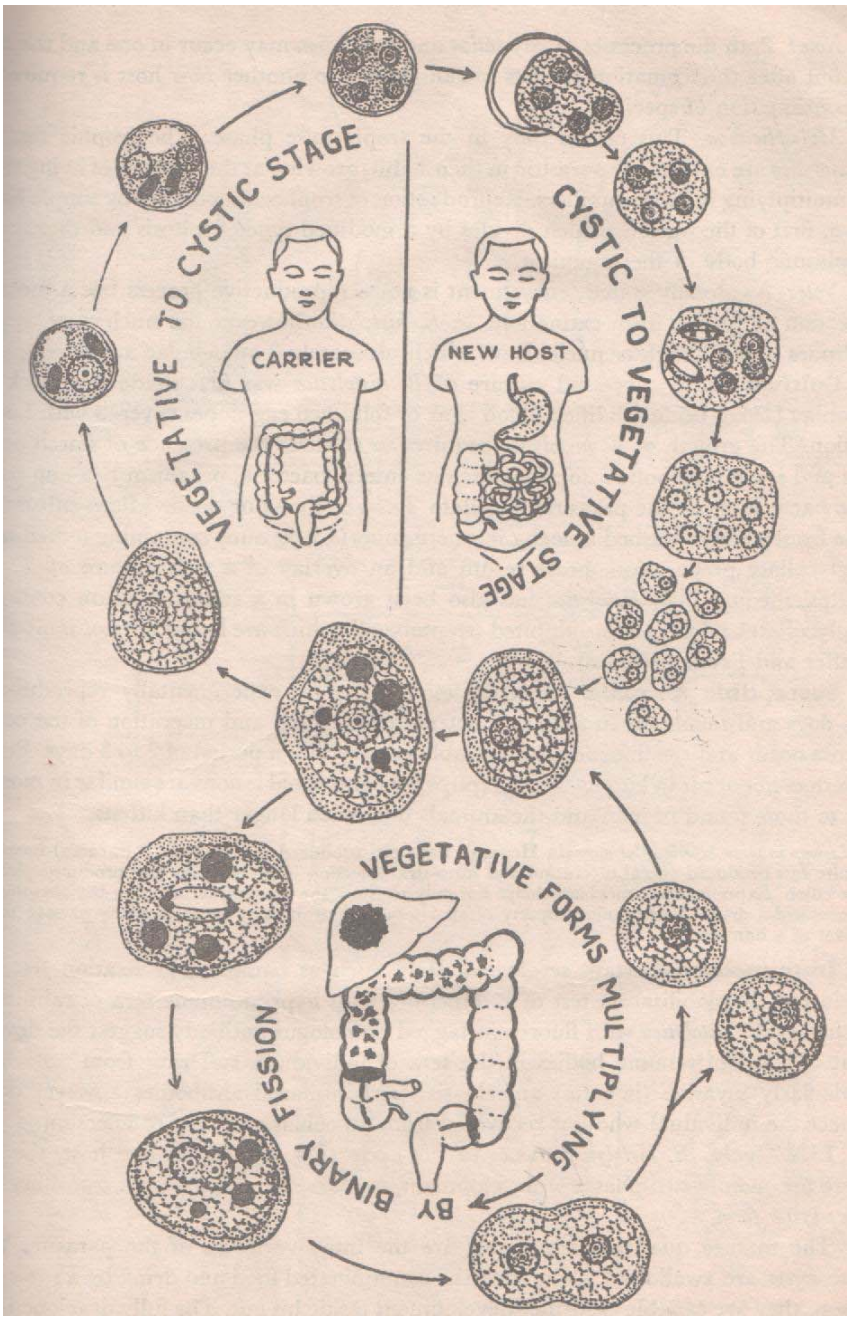
Infection prevalence varies greatly and in some regions exceeds 50%. One study from Gambia, West Africa documented infection rates approaching 100% annually[16].

The new organisms that have been described among the entamoebae species are E. Moshkovskii and E.Dispar .They have been described in Indian population.

The association between amebiasis and warm climates results from the poor sanitation and lack of hygiene that accompany poor living conditions. Infection occurs mainly by the fecal- oral route. Contaminated food, unhygienic handling of food and raw sewage contamination water supplies occasionally causes infection.

"Person-to-person spread as may occur in institutions or in slum areas with large immigrant population accounts for most cases. In occasional patients, no source of infection is evident[17,18,19].

LIFE CYCLE OF E.*HISTOLYTICA*



Microbiology:

The protozoan E. histolytica belongs to the subphylum Sarcodina (whose motility depends on pseudopodia), the superclass Rhizopoda and the order Amoebida. The genus Entamoeba includes the species E. histolytica, E. bartmanni (a non-invasive 'small race' with cysts <10 tm in diameter), E coil, E polecki (infects pigs) and E. moshkovski (a free-living non-pathogenic form found in sewage). Except for E. histolytica, the other species are regarded as non-pathogenic. With the discovery of E.dispar, the identification of E. histolytica on morphology has become unreliable. The presence of ingested erythrocytes is seen only with E. histolytica. The two species have now been characterized by the study of zymodemes (patterns of electrophoretic mobility of isoenzymes) and genetic differences using RNA and DNA probes, and the use of polymerase chain reaction amplification.

E. Histolytica has two forms: Trophozoite and cyst. The Trophozoites are uninucleate, facultative anaerobes with a double-layered limiting membrane surrounded by a fuzzy, external 20-30 nn glycocalyx. With the emerging concepts of virulence, it appears that only certain strains of E. hisiolytica are capable of tissue invasion and contact lysis of cells.

Using the electrophoretic patterns of amoebic enzymes such as glucose-phosphate isomerase, I -malate, NADP oxidoreductase, phosphoglucomutase

and hexakinase, 18 zymodenes of E. histolytica have been described from various areas of the world. Seven of these strains have been isolated from subjects with mucosal ulceration and liver abscess and are consequently labeled as pathogenic.

Cysts of E. histolytica are quadrinucleate. These cysts, measuring 8-20 um, are an important identifying feature, and constitute the infective form of the organism. They are responsible for fecal-oral transmission via food, water or direct person-to-person contact. After ingestion, the quadrinucleate cysts reach the intestinal tract, where they develop into a metacystic stage and undergo an additional nuclear division; thus, eight new uninucleate trophozoites emerge to complete the life cycle. Cysts survive up to 45 minutes in fecal material lodged under the finger nails and up to 1 month in soil at 10°C. They remain infective in fresh water, sea water and sewage but are rapidly destroyed by drying, 200 p.p.m. of iodine and heat above 68°C. They are not killed by chlorination used to purify ordinary drinking water[20].

Host factors

The human host represents the major reservoir although cross-infection from animals - particularly monkeys and rodents has been postulated. Interpersonal transmission occurs via files and handles, and by sewage

contamination of water sources. Male homosexuals also transmit the disease, but usually harbor non- pathogenic E. dispar[21].

A high content of iron in the diet, often obtained from country liquor, predisposes to invasive amoebiasis, and also diet rich in carbohydrate. Young adult males of low socio-economic status are thus the most commonly affected group. Elderly individuals with underlying diseases, and patients with depressed immunity due to malnutrition or corticosteroid therapy, are also prone to invasion by amoebae. The natural resistance of menstruating women is lost in pregnancy.

In Mexican Mestizo population the presence of HLA DR3 and complotype SCO in both adults and children constitutes a primary independent risk factor for the development of amoebic liver abscess, irrespective of age or sex.

Pathogenesis

Entamoeba histolytica exerts a lytic effect on tissue, a characteristic for which the organism is named, Light and electron microscopic studies have been interpreted as showing lysis of mucosal cells on contact with amoeba or alternatively, a diffuse mucosal damage before amoebic invasion. An amorphous, granular, eosionophilic material surrounds trophozoites in tissue, whether in colon, liver, lung or brain. Consistent with the fact those

trophozoites have the capacity to destroy leukocytes; inflammatory cells are found only at the periphery of established amoebic lesions.

Liver pathology in amoebiasis consists of necrotic abscess or periportal fibrosis, the “abscess” contains cellular, proteinaceous debris rather than white cells and is surrounded by a rim of amoebic Trophozoite invading tissue. Amoebae establish hepatic infection by ascending the portal venous system rather than lymphatics. Triangular areas of hepatic necrosis, possibly due to ischemia from amoebic obstruction of portal vessels have been observed. Amoebic liver abscesses probably result from the coalescence of small micro abscesses. Liver function abnormalities are frequently present with intestinal amebiasis and are associated with periportal fibrosis has been reported in such patients; whether this reflects past trophozoites invasion or host reaction to amoebic antigens or toxins is unclear.

Petri et al isolated the E. histolytica gatactose specific adhesion[22]. The adhesin is a 260 -KID surface protein that consists of I7OKD and 35KD subunits. The heavy subunit may mediate attachment as it is recognized by adherence- inhibitory monoclonal antibodies. Direct galactose binding activity of recombinant heavy subunit produced by expression DCR methodology has been demonstrated. The heavy and light subunits are encoded by gene families. The heavy subunit has a short cytoplasmic domain, a transmembrane domain,

and a large extra cellular portion with a distinct cysteine- rich area. The light subunit in contrast is attached to the membrane via a glycosyl-phosphatidylinositol anchor. Petri et a1 identified seven discrete epitopes in the heavy subunit using monoclonal Ig antibodies all of which are located in the cysteine-rich domain.

Entamoeba histolytica contains numerous proteolytic enzymes, including a cathepsin, proteinase, an acidic proteinase, collagenase and a well characterized major neutral proteinase.

Clinical features:

Amoebic liver abscess is 3 to 10 times as common in man as women. Most patients are young adults, although all age groups can be affected. In the more affluent, a history of international travel by the patient to his or her close contacts may be relevant. Specific questions about homosexual activity should be asked. A history of previous dysentery is infrequent and generally unhelpful unless accompanied by dependable laboratory reports.

Symptoms of amoebic liver abscess are slow in onset and usually are present for several days or weeks before medical attention is sought[23]. Initial complaints are vague and include malaise, fever, anorexia and abdominal discomfort. In established cases, pain is most often the dominant symptom and

is more in the right hypochondrium about three fourths of patients complain of fever, often with chills and sweats, particularly at night. Anorexia, nausea and vomiting are common, and many patients lose weight. Chest symptoms are present in about one fourth of patients and include right-sided pleuritic pain and cough. Diaphragmatic irritation may result in right shoulder pain and hiccoughs occasionally. Patients may recognize abdominal swelling. Concurrent intestinal disease, such as dysentery or diarrhea, is rare.

Infrequently, the onset of disease is abrupt and the symptoms mimic those of an abdominal surgical emergency[24]. Sometimes patients complain of ill health for many months, with constitutional symptoms such as weight loss and anaemia predominating. In a small minority of cases, the only manifestation is fever.

On examination , most patients are ill, weak and sweaty, and they may appear anaemic and toxic. Fever and tenderness over the liver are almost invariable; with the tenderness sometimes being most impressive over the right lower intercostals area. Sometimes the liver is visibly enlarged or expands the lower rib cage to give the abdomen an asymmetrical appearance.

Most often the liver is palpable. The physical signs may be subtle when the abscess is in the left lobe of the liver. Presence of epigastric and left

hypochondrial tenderness may arouse suspicion of enlargement of the left lobe of liver.

In about half of the patients, careful examination of the chest reveals abnormalities. Movement of the right side may be limited by pain. Dull on percussion over the right lower lung field is common and implies a raised right hemidiaphragm or pleural effusion. Occasionally there are fine creptations on auscultation or a pleural or pericardial friction rub.

Jaundice is rare and when present, usually of minor degree. It indicates severe illness. Deeper jaundice usually results from multiple or large amoebic abscesses or from lesion situated near the inferior surface of the liver with compression of the larger intrahepatic ducts.

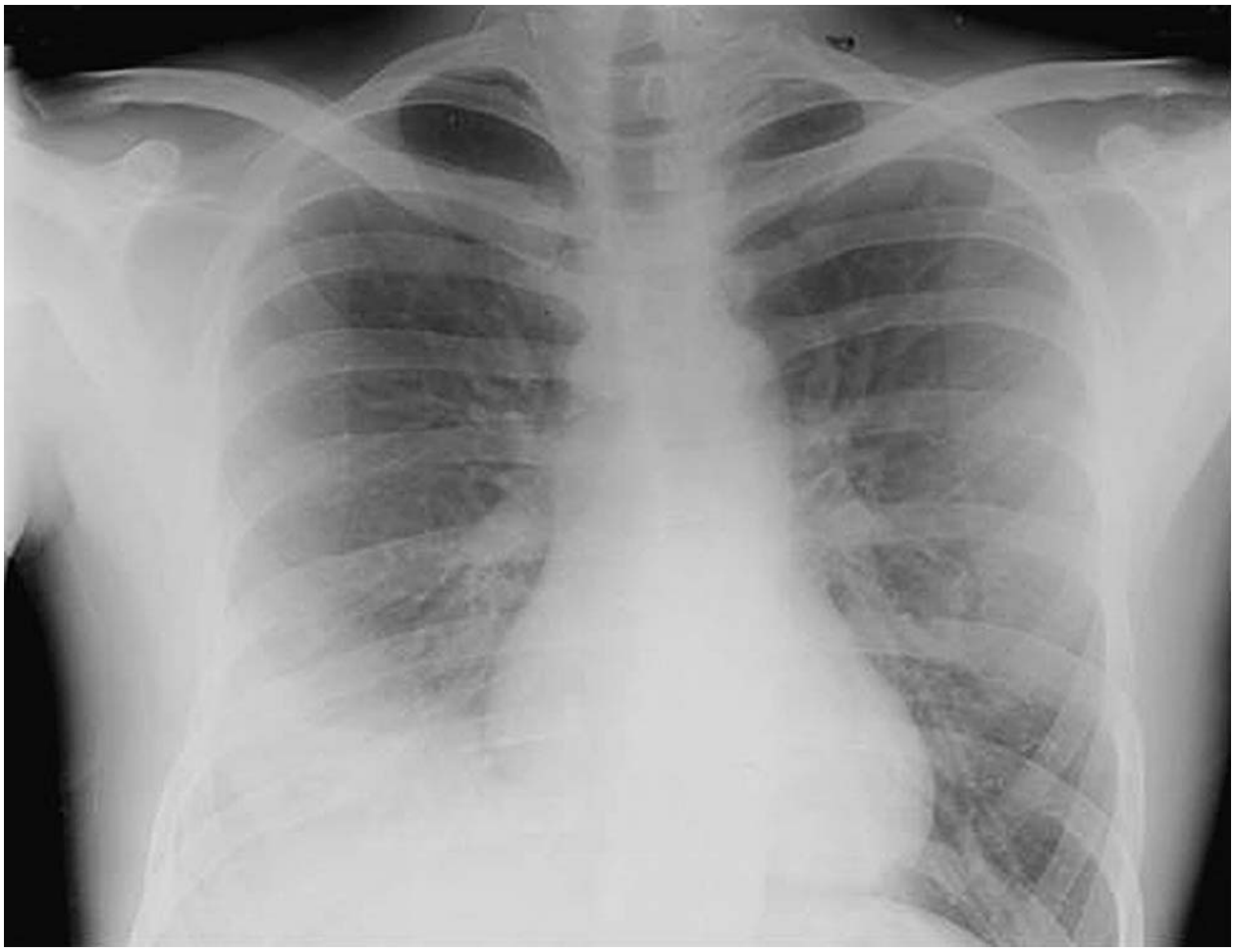
Wherever amebiasis occurs in adults, children may also be infected. Most reported cases of liver abscess in childhood have been in children under age 3, with some affected at only 1 month of life. The sex ratio of cases in children is almost equal. Fever and tender hepatomegaly are the usual physical signs, with the latter sometimes difficult to elicit in crying child. Associated intestinal amoebiasis and multiple hepatic abscesses seem more frequent in children than adults, and malnutrition is an important accompaniment, amoebic liver abscess often seems a severer disease in childhood[25].

When liver abscess occur in pregnancy, frequently such cases are misdiagnosed. A Nigerian autopsy study demonstrated a higher prevalence and mortality from amebiasis in pregnant compared with nonpregnant women. It has been suggested that the immunologic and hormonal alterations of pregnancy predispose to invasive disease. Finally, there is a widespread clinical impression that amoebic liver abscesses is rare in patients with chronic liver disease, although isolated cases have been documented.

DIAGNOSIS

Anaemia is common in amoebic liver abscess, with about half the patients having hemoglobin values below l2gm/dL. Although usually normochromic and normocytic, a hypo chromic blood picture may occur despite adequate iron stores. A neutrophilic leucocytosis is usual and a high proportion of bands may be seen. Although the white blood cell count is between 10,000 and 20,000/ul isolated cases with leukemoid reactions are described. Eosinophilia is not a feature of amebiasis. The erythrocyte sedimentation rate is raised.

Results of liver tests oflen are abnormal and of value in focusing attention of the liver, although derangements may be minor and nonspecific slight elevation of alkaline phosphatase levels and reduction of serum albumin levels are the most frequent abnormal.



Liver abscess rupture into right pleural cavity – right sided pleural effusion

Radiological investigations:

About half of patients show elevation of the right hemi diaphragm of the X-ray chest PA view, the changes in contour being typically most marked anteriorly and medially. Blunting of the right costophrenic angle from a sympathetic pleural effusion is common, as are minor right lower lobe parenchymal abnormalities from atelectasis. Abdominal films may show hepatomegaly but are not helpful. Barium studies and infusion tomography are now outdated techniques for diagnosing amoebic liver abscess.

Technetium sulphur colloid scanning the first modality that allows direct assessment of space occupying liver lesions is sensitive but lacks specificity. Other hepatic masses, such its as tumors and cysts, may produce similar “Cold’ Areas. Gallium scans often are used to complement sulphur colloid scans. Unlike pyogenic abcesses and primary hepatocellular cancers, amoebic abscesses concentrate gallium only at the periphery of the abscess. The disadvantages of these tests include their low specificity, time delay, and the difficulty in working with isotopes[27].

Ultrasonogram is fast, safe, economical, and easily repeatable. A disadvantage of ultrasonography is operator dependency.

Ultrasonic signs mentioned as typical of hepatic amoebic abscess are 1) oval or round shape 2) a lack of notable wall echoes, so that there is abrupt

LIVER ABSCESS – ULTRASONOGRAM

transition from normal liver to the lesion 3) a hypoechoic appearance compared with normal liver, with diffuse echoes throughout the abscess. 4) a peripheral location, usually close to the liver capsule: and 5) a distal sonic enhancement. A typical features that have been documented include on irregular shape and a hyper echoic appearance.

Computed tomographic scanning shows amoebic abscesses well defined, round, low density lesions, which may have a non homogenous internal structure. CT scanning is particularly useful in precise localization and definition of extent of disease (eg, is cases complicated by rupture) Both CT scan and ultrasonography may be used for guidance in cases in which aspiration is indicated. Disadvantages of CT scanning are expensive and the ionizing radiation inherent to the investigation[8,28,29].

Serodiagnosis:

Concurrent hepatic abscess and amoebic dysentery are unusual, stool examination in large series of patients with amoebic abscesses have been negative in three fourths of cases or more. Parasitological examination of the stool specimen can neither prove nor exclude hepatic amebiasis, although it way be relevant for subsequent management. The quality of practical parasitology in hospital laboratories varies widely. Over diagnosis is especially

common, with stools leucocytes frequently reported as trophozoites of E. histolytica.

Serodiagnostic tests used include complement fixation, immunodiffusion, indirect fluoresent antibody tests, indirect hemagglutination (IHA). Counterimmuno electrophoresis, and enzyme — linked immunosorbent assay (ELISA). Commercially produced diagnostic kits for use at the bedside, such as those using latex agglutination are also available. Clinicians should familiarize themselves with local facilities and the accepted sensitivity and specificity of the tests in question.

The IHA test is highly sensitive and widely available. A serologic titer of 1:512 is usual, although not invariable, in acute invasive disease. Titres may continue to rise after presentation, and on occasion, the test is negative when the patient is first seen but positive a few days later. The IHA test may remain positive for months or years after invasive infection. ELISA is a cheap and sensitive technique that has been widely applied to the serodiagnosis and seroepidemiologic study of many parasitic diseases. Its use for the diagnosis of amebiasis is likely to increase.

Role of PCR

Nested PCR and multiple PCR are helpful in differentiations the various species of entameba i.e. E.histolytica .E. dispar., E.meshkorskii[(50)].

Role of aspiration:

In the era before ultrasonography became widely available, aspiration of the typical ‘anchovy sauce’ pus from the liver was often considered vital to confirm the diagnosis of amoebic liver abscess. Nowadays, Ultrasonogram-guided aspiration is often justified on the basis that the diagnosis will then be ‘more certain’ or that the abscess can be ‘aspirate to dryness’ at the time of diagnostic aspiration. The controversy about routine aspiration of uncomplicated amoebic liver abscess remains unresolved. Two recent studies have shown that aspiration does not accelerate healing, and may only confuse the diagnosis by revealing atypical pus or blood. However the belief that aspiration hastens clinical recovery and may not involve significant procedure related morbidity, is widespread in clinical practice. This approach is supported by a recent small prospective study. Clinical improvement invariably occurs with antiamoebic therapy alone in an uncomplicated case. When the differential diagnoses in a given case include operable neoplasm or hydatid disease, aspiration is risky and may even be contraindicated [30,31].

Aspiration is therefore now regarded as generally superfluous in the management of amoebic liver abscess, and should be reserved for situation when

Amoebic liver abscess – USG guided aspiration

Anchovy sauce appearance of amoebic liver abscess pus

I. Amoebic serology is inconclusive, delayed, or unavailable and the main differential diagnosis is a pyogenic liver abscess.

2. A therapeutic trial with antiamoebic drugs is deemed inappropriate (as in pregnancy).

3. There is suspicion of secondary infection of the liver abscess. This is estimated to occur in 15% of cases.

4. When fever and pain persist for more than 3 to 5 days after starting appropriate therapy, aspiration may provide symptomatic relief.

Single aspiration may be sufficient for diagnostic purposes, but when performed as part of therapy is likely to be inadequate. When more than one aspiration is required, the placement of a percutaneous drain is probably indicated to reduce the risk of recurrence.

Complications of amoebic liver abscess:

Communication or extension of amoebic liver abscesses occur into neighboring cavities and organs — the peritoneum, viscera and large vessels on one side of the diaphragm and the pleura, bronchi, lungs and pericardium on the other.

Peritoneal and visceral involvement

Peritonitis associated with amebiasis is due to a rupture of amoebic liver abscess in 78% of cases and due to perforated or necrotizing amoebic colitis in the rest (22%). The two processes can occur simultaneously in a small minority of cases, and this must be kept in mind while making therapeutic decisions for a given patient.

The incidence of spontaneous rupture of amoebic liver abscess varies between 2.7 and 17% of cases. Between 18 and 70% of all amoebic liver abscess ruptures are into the peritoneal cavity. Adherence of the liver abscess to the diaphragm, anterior abdominal wall, omentum and bowel tends to confine the area of contamination. Rupture into a hollow viscus such as the stomach or colon may occur in this situation with spontaneous drainage. A hepatogastric, hepatoduodenal or hepatocolonic fistula may result. Free rupture into the peritoneal cavity is uncommon and usually occurs in a nutritionally depleted and moribund patient. Patients present with abdominal pain, and either a mass or generalized distention. Sudden bloody diarrhea may occur in colonic rupture and hematemesis may occur in patients with hepatogastric fistula. Signs of peritonitis along with tender hepatomegaly, intercostals tenderness and right basal lung signs and clinical jaundice may lead to a suspicion of the diagnosis, which will be confirmed on ultrasonography. At times the diagnosis may be

made only at laparotomy, at which time the excessive bleeding resulting from decreased prothrombin levels can be difficult to manage.

Ultrasonography and CT often show perihepatic fluid collection in cases of amoebic liver abscess. It is not possible by these imaging techniques to tell if these collections are reactive or actual leaks from the abscess cavity, and the differentiation must be made clinically.

Absolute indications for laparotomy include doubtful diagnosis, concomitant hollow viscus perforation with fistulization resulting in life-threatening hemorrhage or sepsis, or if conservative management fails. At laparotomy the liver abscess, which usually appear as a tan-colored bulge on the surface.. Septa running across the cavity are usually blood vessels and bile ducts traversing the abscess cavity. Hemorrhage can be difficult to control, especially if the clotting is disordered and postoperative bile leaks may result. Irrigation of the abscess cavity with saline is usually sufficient and may be followed by the installation for 3-5 mm of a solution of 65 mg of emetine hydrochloride in 100 ml of normal saline. Tube drains are inserted and retained as necessary. Hollow viscus perforations must be dealt with on their own merits, with exteriorization, proximal diversion or serosal patch closure as indicated.

Postoperative antiamoebic therapy in the form of intravenous metronidazole is combined with broad-spectrum antibiotics. Dehydroemetine is added if no cardiac contraindication exists.

Thoracic and pleuropulmonary involvement

Thoracic complications associated with amoebic liver abscess include a sympathetic straw colored right sided effusion, rupture of the abscess into the pleural cavity and rupture of abscess into the bronchial tree.

Transdiaphragmatic involvement in abscess located high on the right lobe is so common as to be part of the clinical syndrome of amoebic liver abscess. Clinically, this manifests as dyspnea and a dry cough which exacerbates the right hypochondrial pain caused by the hepatic lesion. Right basal crepitations are a frequent accompaniment to the abdominal signs. A pleural rub may be found which decreases as sings of pleural effusion supervene. Chest radiography shows atelectasis and blunting of the costophrenic angle. Ultrasonography and CT often pick up the pleural effusion before it is clinically detectable. There are no Ultrasonogram or CT features to differentiate between a sympathetic effusion and a transdiaphragmatic intrapleural rupture except that the former tend to be small and clinically insignificant. No treatment is required for this kind of pleural collection other than the treatment of the liver abscess on its own merits.

Rupture of an abscess into pleural cavity usually occurs suddenly, and extends rapidly to collapse the right lung and fill up the right pleural space. Clinically, it manifests as the sudden onset of severe dyspnea. Radiography reveals a homogenous opacification throughout the right hemi thorax with displacement of the mediastinum to the opposite side. Ultrasonography will reveal the liver abscess.

Treatment consists of thoracocentesis. An important precaution to be observed both in chest aspiration and establishment of intercostal tube drainage is to go in high on the right lateral side of the chest near the axilla, as the right diaphragm is considerably elevated in these patients. The tube can easily be inserted across the diaphragm into the liver, where it will fail to evacuate the pleural collection and may keep the track from the liver abscess from closing. Ineffective early drainage of the amoebic empyema is usually complicated by secondary infection requiring more aggressive surgical procedures like pulmonary decortication at a later date.

Rupture of the abscess into the bronchi is characterized by the sudden onset of coughing with expression of copious quantities of chocolate-colored sputum. Although a complication of amoebic liver abscess, it almost always has a beneficial effect as the abscess drains itself. As the abscess is usually well walled off from both the pleural and peritoneal cavities, surgical intervention is

not required and postural drainage, bronchodilators and antiamoebic drugs suffice. Lung abscess occurs rarely. In cases where adhesions are not well formed, the liver abscess can rupture into both the pleural space and bronchi simultaneously and postural drainage of bronchial secretions must be combined with thoracocentesis.

Metronidazole used as a single drug is effective in the treatment of thoracic complications of amoebic liver abscess, but emetine produces a more rapid response and may be required in cases where metronidazole resistance occurs.

Chemotherapeutic agent

Metronidazole is the treatment of choice for all forms of invasive amoebiasis. It is a nitroimidazole that is well absorbed after oral administration, and it is excreted mainly by way of kidneys.

E.histolytica produces toxic metabolites that interfere with nucleic acid synthesis Adverse effects include nausea,. anorexia, metallic taste, dark urine and a disulfiram like reaction with alcohol. Central nervous system effects such as vertigo, ataxia, and peripheral neuropathy have also been reported. A transient leukopenia occasionally is observed. Reports of possible carcinogenicity, mutagenicity, and the advantages of metronidazole for severe

disease, including in pregnancy, outweigh its, theoretical dangers. In practice, the gastrointestinal adverse effects cause the most trouble[33].

The usual dosage of metronidazole is 800 mg three times daily for 5 to 10 days. The usual paediatric dose is 35 to 50 mg/kg/d in three divided doses. In very ill patients, some clinicians extend treatment for 15 days or even beyond. Patients who cannot take oral metronidazole may be treated with the parenteral preparation. Metronidazole is effective treatment for invasive intestinal disease but is not completely reliable as a luminal amoebicide[34].

Occasional treatment failures have been reported in addition to cases of hepatic amebiasis developing after metronidazole therapy for intestinal disease. Other nitroimidazoles offer no advantage over metronidazole. The best known is tinidazole, which is perhaps associated with less nausea. The usual adult dosage is 2 g/day[35].

Chloroquine: The antimalarial drug chloroquine, a 4- aminoquinoline, acts by binding to parasite deoxyribonucleic acid. High concentrations in liver tissue are obtained after oral administration. It has half life upto 1 week and is excreted predominantly through the kidneys..

Adverse effects are nausea, abdominal discomfort, and pruritis. Retinopathy is only a potential problem in patients taking long term chloroquine, as for malaria prophylaxis or rheumatoid arthritis. The usual dose

is 1 gm / day for 2 days followed by 500 mg/day for 20 days. The only controlled trail of chloroquine versus metronidazole for amoebic liver abscess showed no difference in efficacy other than slightly quicker, response with metronidazole

Emetine and dehydroemetine

Emetine is the oldest as well as the most potent amoebicidal drug available. It is given by intramuscular or subcutaneous injection and slowly excreted through the kidneys. The drug acts by interfering with protein synthesis. The usual dosage is I mg/kg/d to a maximum of 60 mg/day for 10 days. Duration of the treatment should be kept to a minimum, preferably less than 6 days. Adverse effects have rendered this drug obsolete except in the severest of cases. Adverse effects include vomiting, diarrhoea, renal impairment, and pain or necrosis at the site of injection. The most serious adverse effect is cardiotoxicity, any sign of which is an indication for stopping the drug. Emetine has no luminal amoebicidal activity.

Dehydroemetine is a synthetic preparation with a similar action to emetine but associated with less cardiotoxicity. It is equally effective therapeutically but excreted more rapidly. The daily dose of 1.25 mg/kg is given by im or subcutaneous injection to a maximum of 90 mg/d. It should be

given in preference to emetine, if available. Cardio toxicity may be more likely with concurrent administration of chloroquine.

Therapeutic strategy:

Metronidazole is administered as a single drug after diagnosis, with concomitant correction of hypoprothrombinemia, hypoproteinemia, and anaemia. If dramatic improvement in 48 - 72 hours is noted no other therapy other than the complete course of metronidazole is required. A luminal agent such as Dilonaxide furoate (500 mgm p.o. tid x 10 days) or paromomycin (30 mg/kg/day in 3 days x 10 days) must be administered following infection as a part of the complete treatment[34,35,36].

In patients who do not respond satisfactorily, emetine or dehydroemetine is added. Evidence of pulmonary, peritoneal or pericardial extension is all indication for aspiration of the liver abscess with an intercostal tube or catheter drainage into a closed-circuit collection system. Failure to adequately control the abscess by these means — increasing signs or peritonitis, fistulization into a hollow viscus or secondary infection with septicemia constitutes as indications for Laparatomy.

PROGNOSIS

Meta-analysis of 3081 patients with amoebic liver abscess showed that 114 (4%) died. In comparison, the mortality rate for pyogenic liver abscess was

46%. In patients treated with amoebicidal drugs alone the mortality was 2% and the addition of needle aspiration did not improve this result. Independent risk factors for mortality include serum bilirubin more than 3.5 mg%, encephalopathy, hypoalbuminemia less than 2.0 G% and multiple abscess cavities.

Patients treated with a strategy of early and aggressive surgery as advocated by Balasegaram (1981) and Eggleston et al (1982) did not show a remarkable improvement in mortality although in Balasegaram's series the hospital stay was probably reduced[37].

Ruptured amoebic liver abscess occurs in 2-17% of patients, with mortality between 6 and 50%. It is hoped that with increasing skill at percutaneous drainage of these abscesses the mortality in these patients, who usually constitute a major risk for surgery and anesthesia, will be reduced.

MATERIAL AND METHODS

This study was a prospective study done for a period of 2009-2011done on patients in Government Rajaji Hospital , Madurai

Eighty cases of liver abscess were studied during the period

DIAGNOSTIC CRITERIA

All the patients had several investigations required to approach the diagnosis and they were diagnosed as amoebic or pyogenic liver abscess. Basically USG abdomen, serology and pus c/s were done. Serology positive and USG characteristics of smooth wall homogenous with no internal echoes and superficial solitary abscess were grouped as amoebic . Serology negative and pus c/s negative cases with USG characteristics of amoebic abscess were also considered as amoebic abscess

LUNG INVOLVEMENT

X—ray chest PA view was taken in all cases. X—ray findings of right pleural effusion, presence or absence of cough with expectorations were considered as positive.

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TREATMENT SELECTION

Cases with abscess cavity less than 5 cm were treated by drug therapy alone. Failures to relieve symptoms within 3 to 4 days were treated by percutaneous aspiration.

Those with abscess cavity greater than 5 cm were treated either by percutaneous aspiration or by percutaneous catheter drainage. Bilateral abscess cavities that were small and multiple were managed by medical therapy and when any one of the cavity is larger than 5 cm, it was managed by percutaneous aspiration.

Abscess cavities restricted to left lobe were treated by drug therapy if they were multiple and less than 5 cm ; if greater than 5 cm and single were managed either by percutaneous aspiration or by laproscopic drainage.

Those abscess cavities that were larger than 10 cm or with chances of impending rupture in segment III, IV, V, VI were managed by laproscopic drainage.

ADOPTED THERAPEUTIC PROTOCOL MEDICAL

Abscess cavities that were less than 5 cm were treated by Tab. Metronidazole 800 mg for 10 days.

Patient was on i.v metronidazole for three days initially or till the fever subsided. Later oral metronidazole is given and percutaneous aspiration was

done and continued if patient had persisting symptoms after 3 to 4 days of aspiration.

PERCUTANEOUS ASPIRATION

Patient with abscess cavity > 5 cm were treated either by percutaneous aspiration or PCD. Multiple abscesses and the abscess fail to respond with medical treatment were percutaneously aspirated.

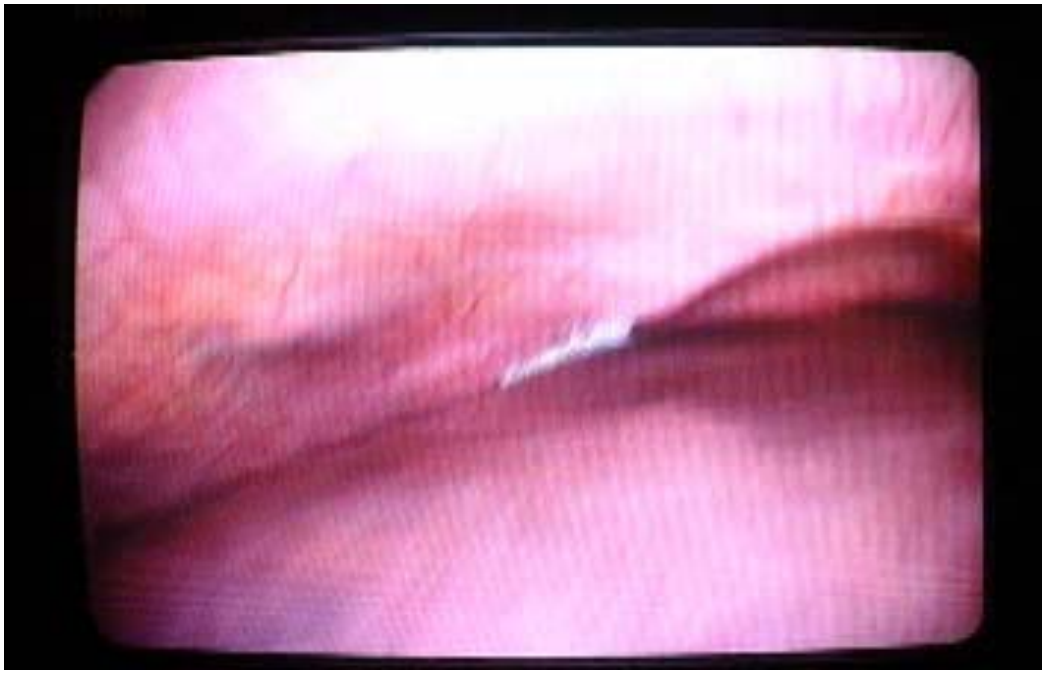
Uunder USG guidance it is done by using 1 6G or 1 8G aspiration needle or 3 way adopter as a single prick. First, aspiration was done followed by drugs. If symptoms are not decreasing after 3 days, do repeat USG and assess the cavity size.

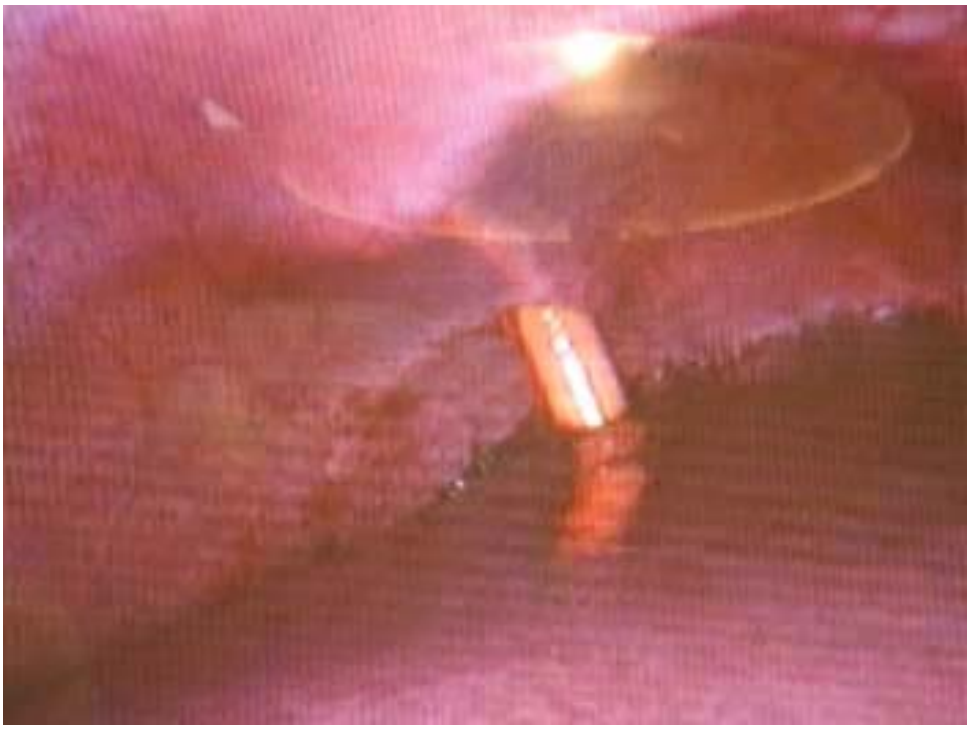
If the cavity is increasing in size or not decreasing do 2^{nd} aspiration and continue drug therapy. If the symptoms are not subsided by 7th postaspiration day and USG showed the cavity is not decreasing or increasing in size, consider PCD or laproscopic drainage.

PERCUTANEOUS CATHETER DRAINAGE

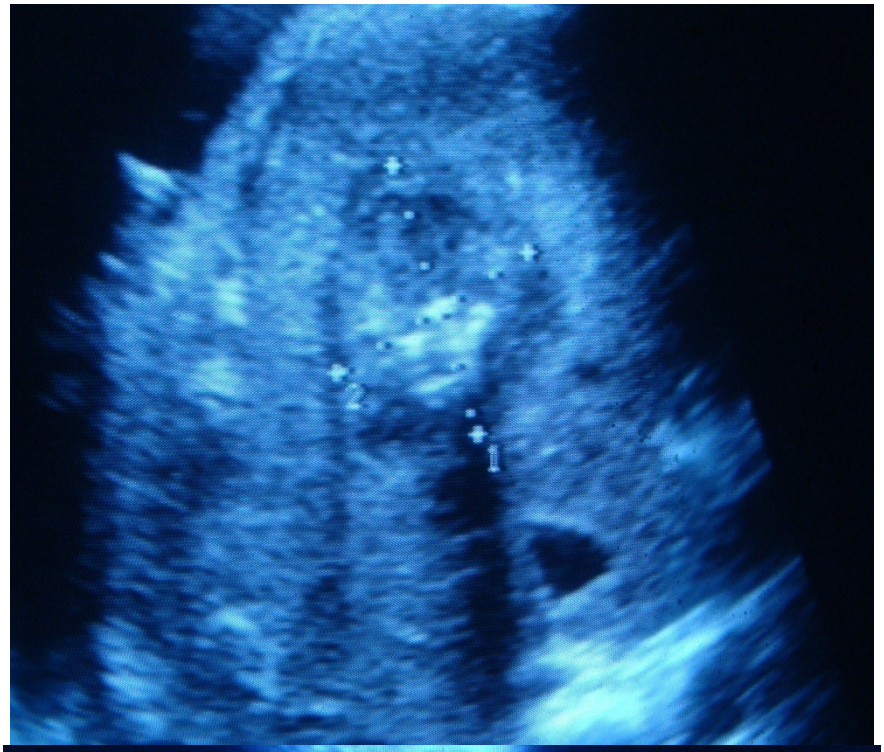
PCD was done by using Malecots /22 F foleys under Ultrasonogram guidance with closed drainage system.

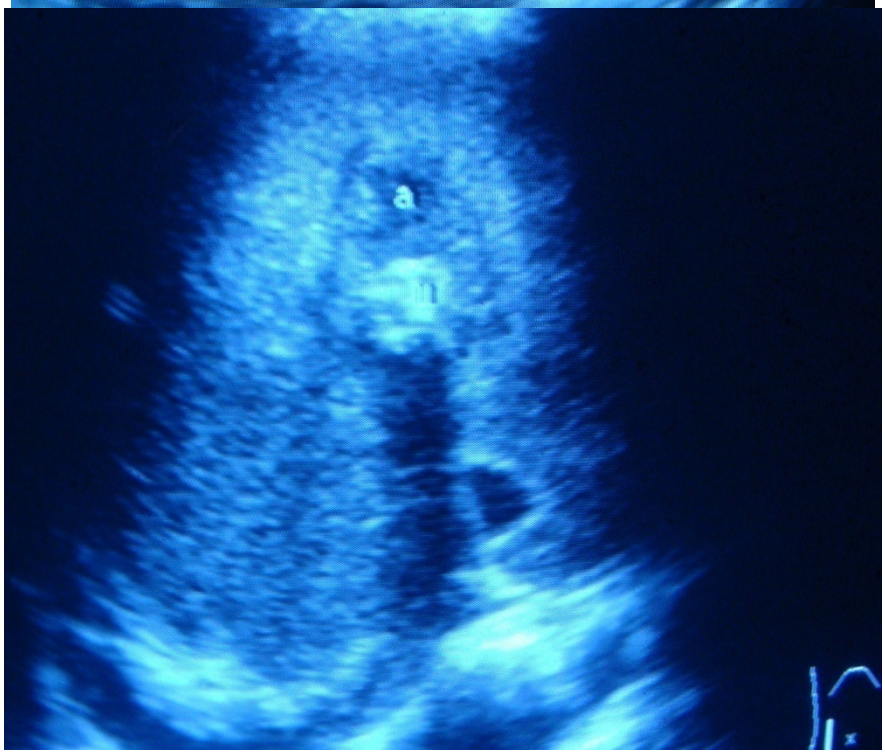
LAPAROSCOPIC DRAINAGE OF LIVER ABSCESS





POST LAPAROSCOPIC DRAINAGE OF LIVER ABSCESS ULTRASONOGRAM FEATURES





PATIENT AFTER POST LAPAROSCOPIC DRAINAGE OF LIVER ABSCESS

REMOVAL OF PCD

1. If the Quantity is less than 30 ml /8hrs.

2. IF the drainage is not purulent.

3. USG and cavitogram were done to assess the cavity size. Note down the decrease in size of the cavity and the PCD can be removed.

LAPROSCOPIC DRAINAGE

Patients with large abscess greater than 10cm and large abscess that was located in the left lobe of liver not amenable to percutaneous drainage were treated by laproscopic catheter drainage. Smaller 16 /14 F foleys used for abscess drainage and the same criteria of removal similar to that of PCD was employed.

RESULTS

The following observations were made in this study

Table 1: Age and sex incidence

Age Group	Male		Female		Total	
	No.	%	No.	%	No.	%
0-30	12	16.66	2	25	14	17.5
31-40	16	22.22	3	37.5	19	23.75
41-50	24	33.33	1	12.5	25	31.25
51-60	15	20.83	1	12.5	16	20
61 yrs	5	6.94	1	12.5	6	7.5
Total	72	100	8	100	80	100

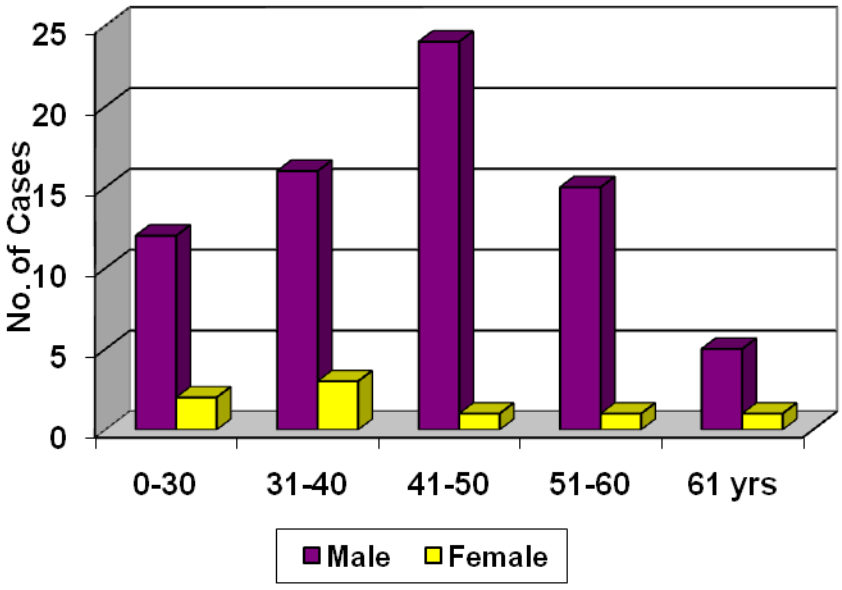


Fig 1. Age and sex incidence

The mean age distribution of the study group is 40.28 ± 7.68 with youngest patient at 19 years of age and oldest patient being 69 years of age.

Liver abscess in this study was more common in males (90%) than females (10%). The commonest age group for liver abscess was 41-50 yrs (31.25%) followed by 31-40 (23.75%).

Symptoms:

Table 2: Incidence of symptoms

Symptoms	No. of patient	%
Fever	57	71.25
Pain abdomen	53	66.25
Jaundice	12	15
Cough	3	3.75
Diarrhoea	14	17.50
Altered sensorium	1	1.25

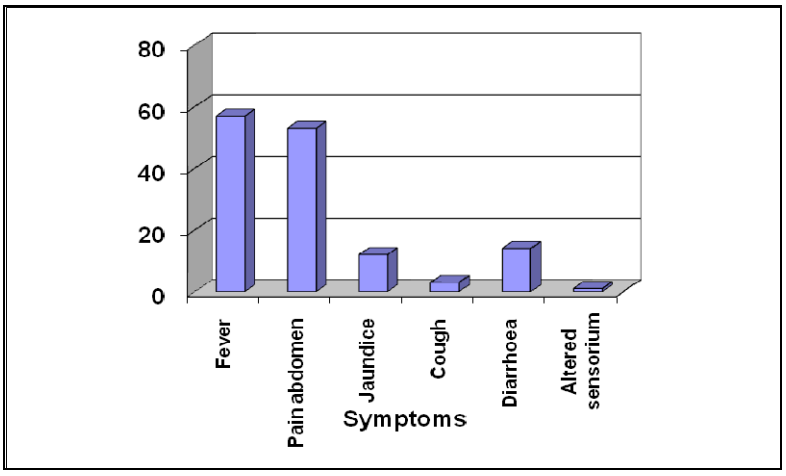


Fig 2. Incidence of symptoms

The commonest symptom was fever(71.25%) followed by pain abdomen (66.25%). Jaundice was present in 15% ,diarrhea occurring in 17.50%, cough in 3.75% and altered sensorium 1.25%.

Table 3: Distribution of signs

Signs	No. of Patient	%
Fever	60	75
Icterus	15	18.75
Pallor	6	7.5
Hepatomegaly	26	32.5
Abdominal tenderness	32	40
Respiratory findings	3	3.75

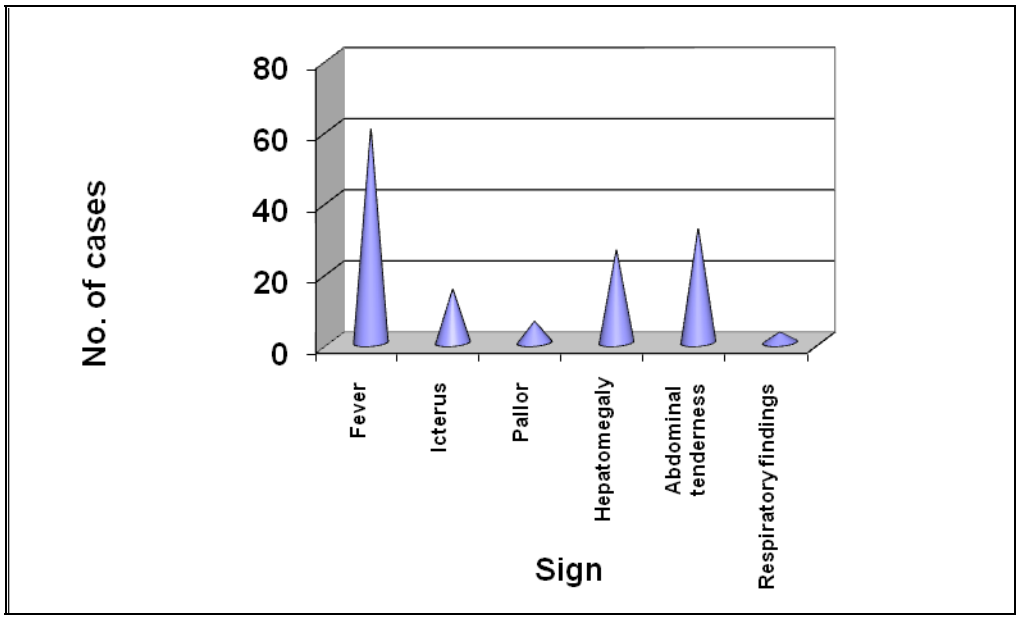


Fig. 3: Distribution of signs

The most common sign was fever which was present in 75% patients, 40% of patients had abdominal tenderness at the time of diagnosis and 32.5% patients had hepatomegaly, 18.75% of patients had icterus, pallor was present in 7.5% of patients and respiratory findings in 3.75% of patients which include right pleural effusion, basal crepitations.

Table 4: Duration of symptoms

Onset	No. of patient	%
Acute < 7 days	42	52.5
Subacute (7 days – 2 months)	19	23.75
Chronic > 2 month	19	23.75

In this study patients presented acutely with onset of symptoms <7 days in 52.5% of cases. Sub acute presentation between 7 days – 2 months was noted in 23.75% of patents and those with chronic duration of onset > 2 months were seen in 23.75% of patients.

Table 5: Alcoholism

Alcoholism	No. of patient	%
Alcoholic	51	63.75
Non alcoholic	29	36.25

Out of 72 males patients, 51 patients were alcoholics. No female patients were found to be alcoholic.

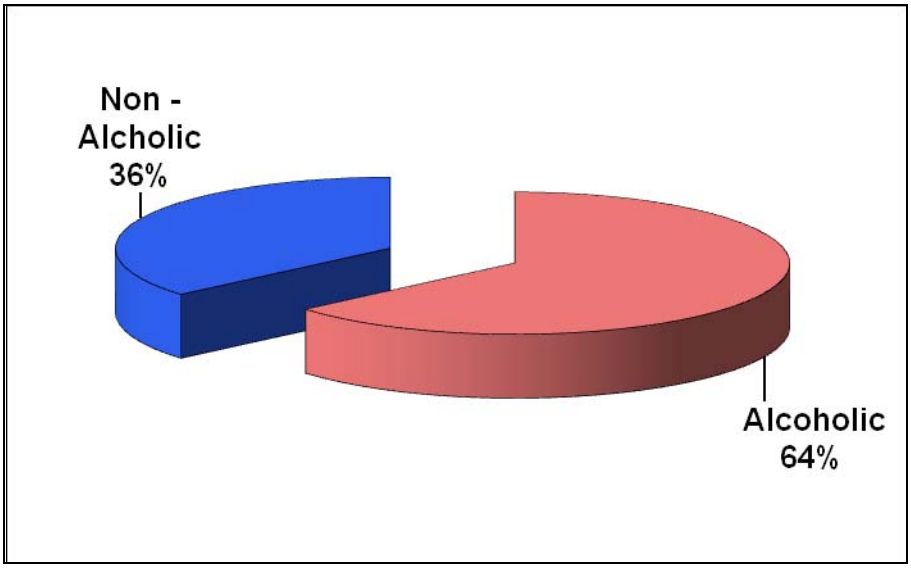


Fig . 4: Alcoholism

Table 6: Percentage of abnormal laboratory investigations.

Investigation	No. of patient	%
Anaemia (Hb < 10 gm%)	8	10
Leucocytosis (> 12,000 c/cmm)	57	71.25
Diabetic (RBS > 200 mg/ dl)	13	16.25
Raised urea (> 60 mg / dl)	9	11.25

Hemoglobin less than 10gm% was found in 8 cases (10%) and lowest hemoglobin noted in this series was 7.6 gm%. Leucocytosis of more than 12,000 cells / cumm was present in 57 patients (71.25%). The highest count noted in this study was 19,000 cells / cumm, 16.25% of patients were found to be diabetic. Raised urea (> 60mg/dl) was found in 9 cases (11.25%).

Table 7 : Analysis of LFT

Serum Bilirubin / mg%	No.of patient	%
< 1	64	80
1.3 – 2	1	1.25
2.1 – 4	9	11.25
4.1 – 6	5	6.25
6.1 – 8	1	1.25

Clinically jaundice was detected in 15 patients with elevated bilirubin levels.

Investigation	**No.of patients**	**%**
ALP (Alkaline phosphatase)	23	28.75
Hypoalbuminemia (< 3gm / dl)	7	8.75
Increased PT time (> 20 sec)	13	16.25
Increased SGOT (> 40 IU/I)	13	16.25
Increased SGPT (> 40 IU/L)	9	11.25

Alkaline phosphatase was found to be raised in 28.75% of cases Hypoalbuminemia (< 3gm/dl) was observed in 8.75% cases. Increased Prothrombin time > 20 sec was seen in 16.25% of cases. Increased SGOT and SGPT were seen in 16.25% and 11.25% of patients respectively .

Table 8 : Chest X-ray

Chest X-ray	No. Of patient	%
Elevated hemidiaphragm	9	11.25
Pleural effusion	3	3.75

Chest X-ray showed elevation of the right hemi diaphragm in 9 cases (11.25%) Obliteration of right costophrenic angle was seen in 3.75% of cases.

Table 9: Ultrasonogram Examination

Ultrasonogram examination was done in all cases. It showed evidence of abscess in liver in all the cases.

Location	No. of patient	%
Right lobe	59	73.7
Left lobe	9	11.25
Both lobes	12	15
Total	80	100

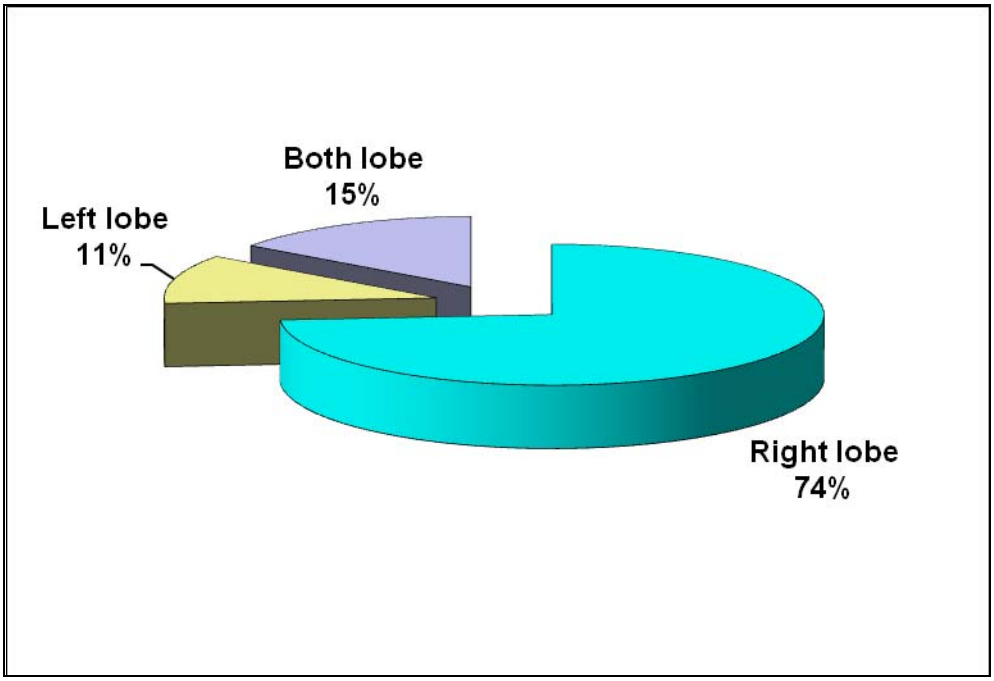


Fig –5 Location of abscess

Right lobe of the liver was more involved than left lobe of liver. Right lobe involvement was present in 73.75% , left lobe in 11.25% and both lobes in 15% of cases .

Table 10: Solitary and multiple abscess

Number	No. of patient	%
Solitary	63	78.75
Multiple	17	21.25
Total	80	100

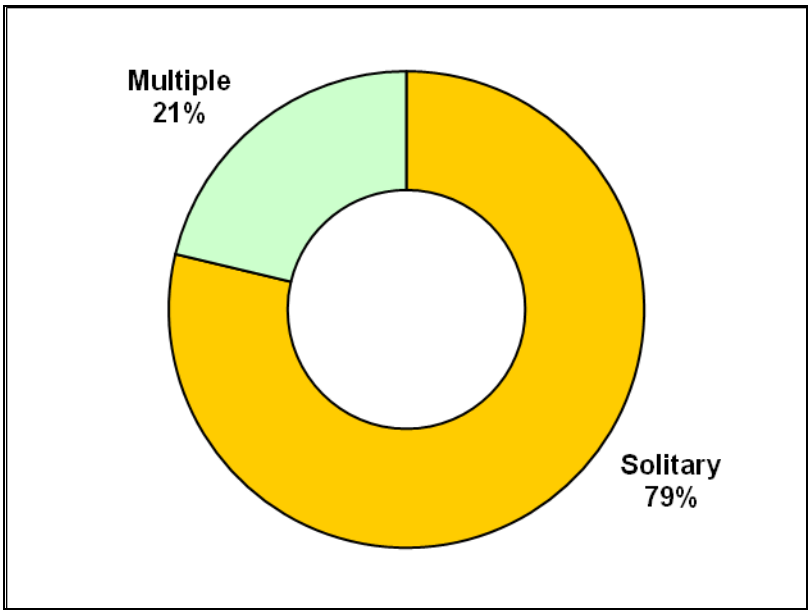


Fig . 6 : Distribution of abscess

78.75% of cases of liver abscess showed solitary abscess in Ultrasonogram examination and 21.25% cases showed multiple abscesses.

Table 11: Pus culture Analysis

Organism	No.of patient	%
No growth / Anchovy sauce	44	84.6%
Non- fermenting gram –ve	6	11.53%
Staph aureus	2	3.84%
Total	52	100%

In this study 52 cases were subjected to invasive treatment. Out of 52 cases, 44(84.6%) had "Anchovy sauce" appearance of the pus and revealed no growth. While growths were obtained in 8 (15.37%) of these cases, non-fermenting gram –ve organisms grown in 6 cases (11.53%) and staph aureus in 2 cases (3.84%).

Table 12: Analysis of treatment

Treatment	No. of patient	%
Conservative	28	35
Aspiration	39	48.75
Laparatomy	6	7.5
Laparoscopic abscess drainage	7	8.75
Total	80	100

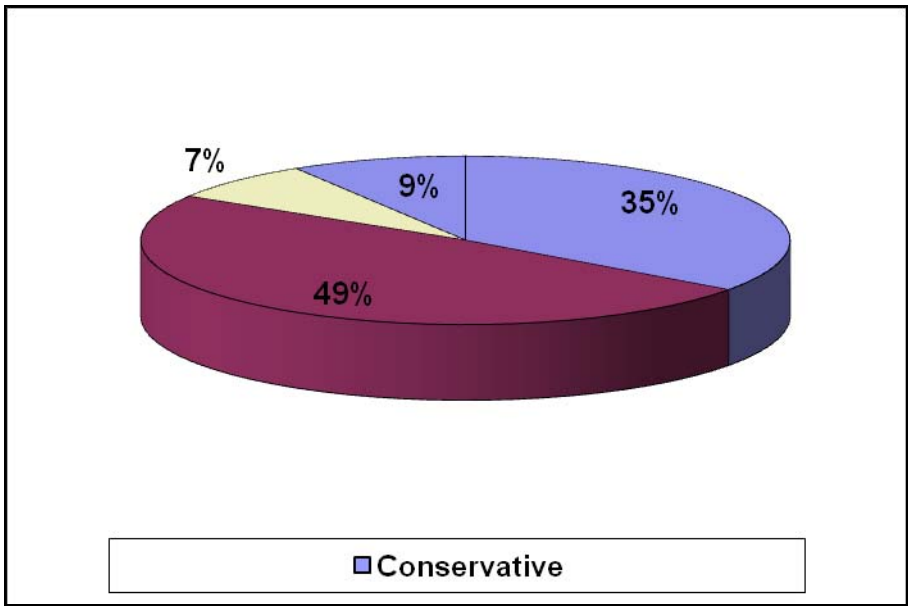


Fig. 7: Treatment of liver abscess

Out of 80 cases, with liver abscess, the volume is < 50 cc is 28 cases (35%) were treated conservatively and those with volume > 50cc were treated by USG guided aspiration is 39 cases (48.75%). 6 cases were treated by Laparatomy and 7 cases by laparoscopic abscess drainage. Abscess ruptured into peritoneal cavity in 6 cases hence laparotomy done. Pus was completely drained out and sent for microbiological examination, peritoneal toilet was given.

Table 13: Complications

Complications	No.of patient	%
Ruptured into peritoneal cavity	6	7.5
Pleural effusion	3	3.75
Septicemia	1	1.25

Table 14: Mortality rate

Total patient with liver abscess	80
Surviving	79
Death due to liver abscess	1

Table 15: Analysis of repeat aspiration

Treatment	No.of patient
Second aspiration	3
More than two aspirations	6

DISCUSSION

Age and sex incidence

The age of the patients varied from 19 – 69 years.. The mean age was 40.28 ± 7.68 yrs. The highest incidence was noted in the age group 41-50 years of age (33. 33%) followed by 31-40 years of age (23.75%) yrs in this study. The mean age was lower when compared to other studies. Bhagwan et al showed mean age of 49 years and the distribution was highest between 41-50 years of age[38] .

• According to Walter D Gaisford James B.D, Mark, the male female ratio is 7:1 whereas in our study we found out that the difference is 9:1 ratio[39].

Table 16: The comparison of symptoms and signs in present study with literature

Symptoms	Our series	Greenstein – et al	Rubin et al
	No of cases (%)	%	%
Fever	71.25	95	87
Pain abdomen	66.25	84	47
Jaundice	15	24	20
Cough	3.75	37	24
Signs			
Fever	75	95	87
Icterus	18.75	24	20
Abdominal Tenderness	40	42	47
Hepatomegaly	32.5	39	51
Respiratory finding	3.75	37	24

Greenstein et al and Rubin et al observed in their studies that fever was present in 95 & 87% of patients respectively whereas in our study we found out a less incidence of fever in 71.25% of patients[40]. Pain abdomen was noticed in 66.25% of patients whereas Greenstein et al. observed a high presentation of pain abdomen in 84% of cases while Rubin et al. observed 47% [41]. Cough was not a significant presentation in our study which was present in 3 patients. Other symptoms and signs were comparable to other studies.

Duration of symptoms

The onset of the disease is subjected to great variations depending upon the type, location and quantity of liver abscess; it may be acute, insidious, clinically undetectable or fulminant form. In this present study acute onset <7 days was seen in 52.5%, sub acute onset 7 days — 2 months was seen in 23.75% of patients and the same 23.75% with the chronic presentation of liver abscess.

According to Bhagwan satiani, Eugene D. Davidson, duration of symptoms prior to admission varied considerably from one day to three months[38].

According to Maingot's abdominal operations, most patients of liver abscess manifest symptoms for less than 2 weeks but a more indolent course occurs in 1/3rd of the patients[42].

Alcoholism in cases of liver abscess

Alcoholism was found to be the most consistent etiological factor in this study of liver abscess. 63.75% of the cases of this study were found to be alcoholics. The presence of alcoholism as a risk factor was noticed in many studies. In Indian culture almost all the alcoholics are males. It can explain the high incidence of liver abscess among the males in comparison to the western literature. According to Shyam Mattur, Alok Mehta et al, the percentage of alcoholics in the patients of liver abscess was noted between 48-71 % of cases[43]. Poor hygiene and eating unhygienic foods were the source of contamination for both the parasitic and bacterial cause of the disease, also plays a significant role in causing liver abscess.

Analysis of laboratory investigations:

10% of patients were found to be anaemic (Hb < 10gm /dl) in our present study. The mean Hb of the patients in this study was 10.4 gm/dl with a range 8.8-13.6 gm%.. According to Bhagwan satiani and Eugene D. Davidson, anaemia was present in 39% of cases[38]. There is less literary evidence suggesting anaemia is a predisposing factor for liver abscess. But

high incidence of anaemia is noted in many of the cases, and the relation is not well understood. Leukocytosis was observed in our cases which was comparable to other studies.

Diabetes Mellitus was observed in 16.25% of patients. The increased association of diabetic state with liver abscess shows that diabetes is a risk factor for liver abscess. According to A.J. Greenstein, D Lowenthal, BA, G.S. Hammer, F. Schaffner and A. H. Aufses, Diabetes was found in 10% of cases[40].

Observations by Bhagwan Satiani and Eugene D.Davidson revealed elevated serum bilirubin was seen in 36% of cases, whereas in our study the elevated bilirubin levels were noted in 19 patients[38].

Raised ALP levels were noted in 28.75% of patients and observations by Bhagwan Satiani and Eugene D. Davidson increased levels of ALP was seen in 63% of cases[38]. According to Chu KM, Fan ST Hypoalbuminemia was an adverse prognostic factor in eases of liver abscess[44]. Increased prothrombin time > 20 was seen in 16.25% increased SGOT in 30% increased SGPT in16.25% of patients.

Chest X-ray finding:

Chest X-ray was done for all patients in this study and the X-ray findings were studied. Chest X-ray showed elevation of the right hemidiaphragrn in 9 cases (11.25%). Obliteration of right costophrenic angle was seen in 3 cases (3.75%).

According to A.J. Greenstein, D. Lowenthal, G,S. Hammer, F. Schaffner and A. H. Aufses, elevation of right hemidiaphragm was seen in 40% of cases and right pleural effusion in 24% of cases[40].

Table 17: USG Findings of liver Abscess

	Present study	Bhagwan satiani et al
	No of cases (%)	
Right lobe	73.75	79
Left lobe	11.25	5
Both lobes	15	16

Ultrasonogram abdomen was done to all patients in this study. In the present study right lobe was involved in 73.75% of cases. This is in accordance with the study concluded by Bhagwan Satiani et al who recorded 79% involvement in right lobe.

In present study left lobe and both lobes were involved in 11.25% and 15 % of patients respectively where as 73. 75% of patients had abscess cavity in the right lobe.

In the present study solitary abscess and multiple abscesses were present in 78.75% and 21.25% of cases respectively. This is in accordance with the study conducted by Chaturbhuj Lal Rajak et a1 who recorded 72% solitary and 18% multiple abscesses[45].

Pus culture analysis

In this study 52 cases were subjected to invasive treatment out of the 52 cases 84% cases had anchovy sauce appearance of pus and revealed no growth . Growth was 15.37% of cases. Non fermenting gram (-) organisms found in 11.53% and Staph aureus found in 3.84%.

Analysis of treatment

Surgical drainage of liver abscesses has been an accepted therapy for decades. The diagnosis and treatment of liver abscess has changed due to advances in imaging techniques.

In the present study of 80 cases patients who had multiple small abscess and solitary abscess with volume <50 ml were treated conservatively. The conservative management was done on 35% of cases.

All cases were started on metronidazole IV at a dose of 40 mg/kg/wt (2.0—2.5gm/day in divided doses for 8-10 days). When patients did not show improvement in 24-48 hrs of metronidazole therapy, broad spectrum 3rd generation cephalosporins were started.

According to Hiroshi Okano, Katsuya Shraki percutaneous aspiration is not required in all cases of liver abscess. A subset of cases with small liver abscess < 300 cc can be successfully managed conservatively[46].

In 48.75% patients who had abscess >50 cc were chosen for percutaneous aspiration. The site, depth and direction of aspiration were marked under USG guidance, aspiration needle was usually used and under aseptic precautions, the abscess cavity was entered. Local anesthetic was used, pus was aspirated and sent for culture and sensitivity; no complication were noted due to this procedure apart from local pain which soon subsided after analgesics. Patient showed improvements in their symptoms and signs within 48-72 hrs of the aspiration. Percutaneous catheter drainage was not done on any patient in this study. Laparotomy as the initial line of treatment was performed in 6 (7.5%) of cases of liver abscess ruptured into peritoneal cavity. On laparotomy, thorough peritoneal lavage and drains were kept.

According to Arshed Zafar, Sajjad Ahnied, needle aspiration is safe, rapid effective method of treating liver abscess. Routine aspiration is not indicated. It should be initial line of treatment in abscess > 300 cc, impending rupture or abscess that do not respond to chemotherapy.

According to Antonia, Giorgio, Lucien Turuntino percutaneous needle aspiration is an efficient, effective and low cost technique that can even be performed on an out patient basis [48]. It is safe, free from significant complication.

Laparatomy was done in 6 cases for liver abscess which ruptured intraperitoneally. Laparoscopic liver drainage was done in 7 patients which is technically difficult for the young laparoscopic surgeons.

Analysis of Repeat Aspirations

A Single repeat aspiration was sufficient in 3 patients while in 6 patients, multiple repeat aspirations were required. Thick viscous pus was the main reason for repeat aspirations.

According to Chaturbhuj Lal et al, in their study on liver abscesses found that single aspiration was successful in 88% of cases while 10% required 2 aspirations[45].

Discussion of complications

The complications in our study were rupture of liver abscess into peritoneal cavity and pleural effusion. Six case presented with peritonitis for which laparotomy was done and peritoneal lavage was given. Septicemia with multiorgan dysfunction was seen in 1 case. Pleural effusion was observed in 3 cases.

According to Sharma MP, Dasarthy S, Verma N et al, mortality rate in their study was 0-18 % and in our study one case (1.25%) had the end result of mortality due to liver abscess after intraperitoneal rupture[49].

CONCLUSION

This study is based on the reports of 80 patients treated for liver abscess at Govt. Rajaji Hospital, Madurai.

- The most common age group affected by Liver abscess was between 41-50 years.
- The male-female sex ratio found in this study was 9:1.
- The most common symptom was fever, followed by pain abdomen.
- Among the risk factors studied, alcohol consumption was observed in the most number of patients.
- Solitary abscesses were common compared to multiple abscesses.
- The right lobe was more commonly affected.
- 2/3 of the patients needed invasive management.
- Multiple small abscesses and solitary abscess with volume less than 50 ml were managed successfully on conservative antimicrobial therapy alone.

- Complications like intraperitoneal rupture, pleural effusion septicemia, and death can occur. Mortality rate in this study was 1.25%.

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ANNEXURE

PROFORMA

Patient's Name :	Address :
Age :	Hospital :
Sex :	Ward No :
Occupation :	Unit :
Locality : Urban / Rural	D.O.A :
Religion :	D.O.D :
Duration of stay in hospital :	Socio economic status:

CHIEF COMPLAINTS :

HISTORY OF PRESENTING ILLNESS:

Symptoms

1. **Abdominal Pain : Yes / No**
    - Duration :
    - Site :
    - Character :
    - Radiation :

2. **Fever : Yes / No**
    - Duration :
    - Type :
    - Associated with chills and rigors :
3. **Diarrhoea / Dysentery :**
    - Duration :
    - Mucous diarrhea :
    - Blood in stools :
4. **Vomiting :**
    - Duration / Frequency :
    - Color :
5. **Jaundice : Yes / No**
6. **Cough : Yes / No**
7. **Distension of abdomen: Yes / No**
8. **Altered Sensorium : Yes / No**
9. **Any Other :**

PAST HISTORY

Diarrhea : Yes / No

Jaundice : Yes / No

Diabetes : Yes / No

Tuberculosis : Yes / No

Surgery : Yes / No

Trauma : Yes / No

FAMILY HISTORY :

Similar illness :

Other :

PERSONAL HISTORY :

ALCOHOL CONSUMPTION:

Duration	
Amount	

GENERAL PHYSICAL EXAMINATION :

Pallor :

Jaundice :

Respiration : per minute

Temperature :

LOCAL EXAMINATION :

ABDOMEN:

INSPECTION :

1. Shape : Scaphoid / flat / distended / obese

PALPATION :

1. Tenderness : Present / Absent

2. Rigidity : Present / Absent

3. Liver : Palpable / non palpable

Upper limit of percussion in rt MCL : LCS

Lower limit of percussion below costal margin: cms

Liver span : cms

Borders : sharp / rounded

Surface : Smooth / Nodular

RESPIRATORY SYSTEM :

1. Shape and expansion of chest :
2. Air entry :
3. Breath sounds :
4. pleural effusion : Present / Absent

INVESTIGATIONS:

1. **Blood :**

Hb : gm%

T.C. : c/cmm

D.C. : N%, L%, E%, M%, B%

ESR : mm/hr

Prothrombin time : Sec

INR :

FBS : mg%

Blood urea : mg %

SPECIAL INVESTIGATIONS:

Liver Function Tests:

1. Serum Bilirubin : mg%

2. Albumin : gm%

3. Alkaline phosphatase : IU/L

4. S.G.O.T. IU/L

5. S.G.P.T. IU/L

6. PT : IU/L

RADIOLOGICAL INVESTIGATIONS:

1. Plain X-ray Chest PA view :
2. **ULTRASONOGRAPHY ABDOMEN :**

TREATMENT :

MEDICAL TREATMENT:

SURGICAL TREATMENT

COMPLICATIONS :

Early :

Late post operative :

FOLLOW UP :

COMMENTS:

KEY TO MASTER CHART

-	-	Absent
+	-	Present
ALP	-	Alkaline Phosphatase
B	-	Both lobes
CxR	-	Chest X-ray
DUR	-	Duration
EHD	-	Elevated hemi diaphragm
Hb	-	Hemoglobin
L	-	Left lobe
M	-	Multiple abscess
NAD	-	No abnormal defects
PR	-	Peritoneal rupture
PT	-	Prothrombin time
RBS	-	Random blood sugar
RPE	-	Right pleural effusion
R	-	Right lobe
S	-	Single
S. Alb	-	Serum Albumin
WBC	-	White blood count

											MASTER CHART													
S.No	IP No.		Sex	Abdominal Pain	Fever	Duration(days)	Alcoholism	Icterus	Hepatomegaly	Hb%	WBC	RBS	Urea	S.bilurubin	ALP Raised	S.Alb(<3gm/dl)	SGOT(>40IU/ml)	SGPT(>40IU/ml)	PT(>20s)	Chest Xray	Lobe Involved	Single/Multiple	treatment	Complication	Recurrence	Pus C&S
1	O22825	32	M	+	+	67	+	-	Yes	10	11,000	80	23	O.3	Yes	No	Yes	Yes	No	EHD	R	S	Conservative	Nil	No	No grwth
2	O25626	41	M	-	+	4	-	-	No	10.4	12,500	254	16	4.2	No	No	No	No	No	NAD	R	S	Conservative	Nil	Yes	No grwth
3	O25815	55	M	+	-	3	+	+	No	9.6	13,000	70	28	O.4	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
4	O32274	35	M	-	-	3	-	-	Yes	11	13,500	90	15	1.6	Yes	Yes	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
5	O35800	23	M	+	+	87	-	-	No	11.2	13,000	98	70	O.6	No	No	Yes	No	Yes	NAD	B	M	Conservative	Nil	No	No grwth
6	O38902	23	M	+	-	14	+	-	No	10.3	12,500	106	29	O.7	No	No	No	No	No	NAD	R	S	Aspiration	nil	No	Non Ferm Gm-ve
7	O42131	36	F	+	+	6	-	+	Yes	11	8,000	227	26	O.4	No	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
8	O45550	47	M	-	+	4	+	-	No	10.3	19,000	105	130	7.4	Yes	No	Yes	Yes	Yes	NAD	R	S	laparotomy	Septicemia	No	No grwth
9	O47201	49	M	+	-	45	+	-	No	9	10,500	140	69	0.4	No	No	No	No	No	EHD	R	S	Conservative	Nil	No	No grwth
10	O56522	39	M	-	+	76	+	+	Yes	10.8	11,000	120	19	2.1	No	No	Yes	No	Yes	RPE	R	M	laparotomy	PR	No	No grwth
11	O69585	48	M	+	-	3	+	-	No	11.3	13,500	110	29	O.5	Yes	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
12	O71212	37	M	-	+	4	-	-	Yes	10.5	13,000	89	19	O.6	No	Yes	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
13	O53356	52	M	+	+	23	+	-	No	11.6	12,800	270	29	O.3	No	No	Yes	No	No	NAD	R	M	laparotomy	Peritoneal rupture	No	No grwth
14	O56264	44	M	+	-	4	+	-	No	11.2	12,400	85	74	2.5	No	No	No	Yes	No	NAD	R	S	Aspiration	Nil	No	No grwth
15	O62331	35	M	+	+	85	-	-	Yes	10.4	13,000	96	10	O.5	Yes	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
16	O62472	31	M	-	+	42	-	-	No	10.6	13,500	86	14	O.6	No	No	Yes	No	Yes	EHD	R	S	Conservative	Nil	Yes	No grwth
17	O69302	25	M	+	+	67	+	-	No	10.2	9,000	89	30	2.8	Yes	Yes	No	Yes	No	NAD	L	M	laparoscopy	Nil	No	No grwth
18	O69562	46	M	-	+	5	-	-	Yes	10.4	10,500	110	33	O.5	Yes	No	No	No	No	NAD	B	S	Conservative	Nil	No	No grwth
19	O91970	33	M	+	+	13	+	+	No	10	12,600	120	20	O.6	No	No	No	No	Yes	NAD	R	S	Conservative	Nil	Yes	No grwth
20	O94831	27	F	-	-	5	-	-	No	9.8	12,800	70	29	2.7	No	Yes	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
21	O95717	44	M	-	+	87	-	+	No	10.1	13,600	98	15	O.7	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
22	O105374	34	M	+	+	13	+	-	Yes	10.3	7,000	220	18	O.7	No	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
23	O11427	45	M	+	+	5	+	+	No	10.5	13,500	79	16	2.3	Yes	No	No	No	No	NAD	R	M	laparotomy	PR	No	No grwth
24	O27720	58	M	+	-	4	+	-	No	9	14,000	280	29	O.7	No	No	No	No	No	NAD	B	S	Conservative	Nil	No	No grwth
25	O67546	18	M	-	+	6	+	-	Yes	11.2	12,800	86	19	3.2	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
26	O75661	33	M	+	+	33	-	-	No	10	11,000	160	29	O.4	No	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
27	O80098	42	M	+	-	64	+	+	Yes	10.4	13700	80	55	2.9	Yes	No	No	No	No	NAD	R	S	laparoscopy	Nil	No	No grwth
28	O10190	28	M	+	-	36	+	-	No	11.4	13,500	78	29	O.4	No	No	No	No	No	NAD	B	S	Aspiration	Nil	No	No grwth
29	O78212	57	M	-	+	4	-	-	No	11	9,000	104	29	O.7	No	No	Yes	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
30	O79687	37	M	+	+	97	+	+	No	10.4	12,500	110	69	O.4	Yes	No	No	No	No	EHD	R	S	laparoscopy	Nil	Yes	No grwth
31	O91966	19	M	+	-	6	+	-	No	12	13500	120	29	O.7	No	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
32	OO9170	46	M	+	+	76	+	-	Yes	10.3	11000	140	19	O.2	Yes	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth

33	O10812	31	M	+	+	6	-	+	No	10.4	14000	90	39	O.8	No	No	Yes	No	Yes	NAD	R	M	Aspiration	Nil	No	No grwth
34	O13989	56	M	+	-	34	+	-	Yes	10.6	14500	254	25	O.5	No	No	No	No	No	NAD	L	S	laparoscopy	Nil	No	No grwth
35	O14006	29	M	+	+	5	+	-	No	8	13600	140	19	O.7	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
36	O29981	35	M	-	+	87	-	+	No	10.2	13900	120	15	O.8	No	No	No	No	No	RPE	R	S	Aspiration	Pl. Eff.	No	Staph. Aureus
37	O30024	48	M	+	+	4	+	-	Yes	10.6	12900	110	30	O.5	No	No	No	No	No	NAD	L	S	laparoscopy	Nil	No	No grwth
38	O52541	59	M	+	-	67	+	-	No	12	15500	324	20	O.6	Yes	No	No	Yes	Yes	NAD	L	S	Conservative	Nil	No	No grwth
39	O16068	39	M	-	+	4	+	+	No	11.5	9000	124	29	O.4	No	No	No	No	No	EHD	R	M	laparoscopy	Nil	No	No grwth
40	O46813	45	M	+	-	12	+	-	Yes	10.6	13000	140	35	O.3	No	No	No	No	No	NAD	R	S	Aspiration	Nil	Yes	No grwth
41	O46919	51	M	+	+	4	+	-	No	10.4	14000	80	43	O.5	Yes	No	No	No	No	NAD	B	S	Aspiration	Nil	No	No grwth
42	O51487	25	M	+	+	4	-	-	No	11	13700	68	43	O.2	No	No	Yes	No	Yes	NAD	L	M	Aspiration	Nil	No	No grwth
43	O62779	54	M	-	-	5	+	-	No	11.8	8600	89	49	O.7	No	No	No	No	No	NAD	R	S	laparotomy	PR	No	No grwth
44	O64826	42	M	+	+	88	+	-	Yes	12.2	13100	324	39	4.5	No	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
45	O67502	57	M	-	-	6	-	+	No	10.4	12000	89	29	O.5	Yes	No	No	No	No	NAD	B	M	Aspiration	Nil	No	No grwth
46	O69096	37	M	+	+	3	+	-	Yes	10.9	12300	230	20	O.5	No	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
47	O62977	26	M	+	+	43	+	-	Yes	10.4	15000	114	40	O.2	No	No	Yes	No	Yes	NAD	L	S	Aspiration	Pl. Eff.	No	No grwth
48	O13799	49	M	-	+	87	-	-	No	7.8	7000	108	22	5.4	Yes	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
49	O16885	54	M	+	-	67	+	-	No	10.4	12000	78	21	O.6	No	No	No	No	No	NAD	R	S	laparoscopy	Nil	Yes	No grwth
50	O32180	45	M	+	+	4	+	-	Yes	10.5	10000	77	90	O.7	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	Non Ferm Gm-ve
51	O48824	36	M	+	-	12	+	-	No	11	15200	209	30	O.6	No	No	No	No	No	NAD	R	M	Conservative	Nil	No	No grwth
52	O53118	52	M	+	+	6	-	-	No	11.2	11000	89	36	O.4	Yes	No	Yes	No	Yes	EHD	R	S	Aspiration	Nil	No	No grwth
53	O55240	43	M	+	+	87	+	-	Yes	11.5	12000	97	82	O.3	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
54	O56435	62	F	+	+	3	-	+	No	12.4	12500	250	39	O.8	No	No	No	No	No	NAD	B	M	Conservative	Nil	No	No grwth
55	O59224	41	M	-	+	31	+	-	No	11.7	12400	115	18	O.9	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	Non Ferm Gm-ve
56	O62519	66	M	+	-	3	+	-	Yes	10.5	10050	140	34	3.6	Yes	No	No	No	No	NAD	R	M	Aspiration	Nil	No	No grwth
57	O11380	47	M	-	+	78	+	-	No	7.8	14000	280	14	O.3	No	No	No	No	No	EHD	R	S	Conservative	Nil	No	No grwth
58	O12456	64	M	+	+	34	+	-	No	11.3	15000	224	19	O.5	No	No	No	No	No	NAD	L	S	Aspiration	Nil	No	No grwth
59	O41311	26	F	-	+	4	-	-	Yes	11.4	11000	110	33	O.6	No	No	Yes	No	Yes	NAD	R	S	laparotomy	PR	No	No grwth
60	O48210	20	M	+	+	4	+	-	No	10.2	12500	105	39	O.4	Yes	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
61	O48197	22	F	+	+	6	-	-	No	10.5	10500	144	68	O.6	No	No	No	Yes	No	NAD	L	S	Aspiration	Nil	No	No grwth
62	O49717	57	M	+	+	34	+	-	Yes	12.8	11000	117	20	O.7	No	No	No	No	No	NAD	R	M	Conservative	Nil	Yes	No grwth
63	O39872	39	M	-	+	6	15200	-	No	11.3	15500	105	69	O.2	No	Yes	No	No	No	NAD	R	M	Aspiration	Nil	No	No grwth
64	O49724	49	M	-	+	54	+	-	No	10.5	12000	98	59	O.6	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
65	O49899	69	M	-	-	3	+	-	Yes	10.2	13000	154	26	O.5	No	No	No	No	No	NAD	B	S	Conservative	Nil	No	No grwth
66	O32199	57	M	+	+	6	+	-	No	10.8	15000	86	39	O.7	No	No	No	No	No	NAD	B	S	Aspiration	Nil	No	Non Ferm Gm-ve
67	O48834	22	M	-	+	3	-	-	Yes	]11.6	14500	69	25	O.8	Yes	No	No	No	No	EHD	R	M	Aspiration	Nil	No	No grwth
68	O53418	42	M	+	+	3	+	+	No	11.9	12500	89	53	O.6	No	Yes	No	No	No	NAD	B	S	Conservative	Nil	No	No grwth
69	O55540	32	F	+	+	97	-	-	No	13.4	15000	104	22	5.2	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
70	O56635	59	M	+	+	3	-	-	Yes	13.5	13000	114	25	O.4	No	No	Yes	No	Yes	NAD	R	M	Aspiration	Nil	No	No grwth
71	O59264	68	M	-	-	1	-	-	No	10.2	11300	189	30	O.6	No	No	No	Yes	No	RPE	B	S	Conservative	Nil	Yes	No grwth

72	O62523	43	M	+	+	78	+	-	No	10	12600	164	10	4.8	No	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
73	O11450	47	F	-	+	26	-	-	Yes	10.3	12050	112	16	O.7	Yes	No	No	No	No	EHD	R	S	Conservative	Nil	No	No grwth
74	O12786	52	M	+	+	4	+	-	No	10.5	13000	135	19	O.8	No	No	No	No	No	NAD	B	S	Aspiration	Nil	No	No grwth
75	O41671	41	M	-	-	2	+	-	No	10.5	14300	96	34	1.9	No	Yes	No	Yes	Yes	NAD	R	S	Aspiration	Nil	Yes	No grwth
76	O48910	36	F	+	+	3	-	-	No	11.2	13500	92	20	O.4	Yes	No	No	No	No	NAD	R	S	Aspiration	Nil	No	No grwth
77	O48168	56	F	-	-	76	-	-	No	11.5	12400	105	39	O.6	No	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
78	O49734	45	M	+	+	34	+	+	Yes	11	15000	78	28	O.8	No	No	No	No	No	NAD	R	S	Conservative	Nil	No	No grwth
79	O39884	38	F	+	+	87	-	-	No	7.6	12500	139	20	O.7	Yes	No	No	No	No	NAD	R	M	Aspiration	Pl. Eff.	No	No grwth
80	O45566	46	M	-	-	3	+	-	No	10.7	13000	129	28	O.7	Yes	No	No	Yes	No	NAD	L	S	Aspiration	Nil	No	No grwth